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URETERIC INJURIES IN GYNAECOLOGY

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According to Ingersoll and Meigs[1] 'accidental injury to the ureter is the most serious complication of gynaecological pelvic surgery'. While there must be general agreement on the seriousness of this complication, it is almost impossible to obtain any reliable figures of its incidence, because a very definite proportion pass unnoticed, giving rise to minimal postoperative symptoms, and are, in fact, discovered only in the postmortem room. Newell[2] reports 6 such cases of ureteric ligation found at autopsy, none of which was even suspected before this examination. Many more such cases have since been recorded.

In a series of 857 hysterectomies performed in London at the Samaritan Hospital between 1952 and 1954, Ostry[3] reports injury to 6 ureters, an incidence of 0·7%. Similarly, over a period of 5½ years, Conger *et al.*[4] discovered 13 ureteric injuries in just under 2,400 major pelvic surgical operations at the Temple Hospital in Philadelphia — a percentage of 0·56. The most carefully controlled series is that of St. Martin,[5] who carried out routine pre- and postoperative intravenous pyelograms on all of his cases, where necessary cystoscoping and doing retrograde pyelograms between the 6th and 18th postoperative days. In his total of 332 cases he found evidence of ureteral injury in 8 cases (or 2·42%). While these figures are far higher than in the previous series quoted, due allowance must be made for the fact that 5 of St. Martin's 8 cases, considered to be due to minor trauma of the lower ureter, had only temporary hydronephrosis and showed complete restoration to normal, without any treatment, by the 5th week after surgery. Of 3 cases of complete ureteric ligation in his series, 2 were completely asymptomatic. From the same hospital 6 of 20 cases of Wertheim hysterectomy for carcinoma of the cervix showed ureteric injury. Though this figure is far higher than might be anticipated, it is indicative of the high incidence of trauma to ureters in radical pelvic procedures. Lui and Meigs,[6] analysing a series of 473 radical operations from a group of Boston hospitals, found 45 ureterovaginal fistulae, i.e. 9%. As it is common knowledge that only a proportion of ureteric injuries go on to fistula formation, the actual incidence of ureteric injuries in this series must have been much higher.

Anatomy

In a lecture at the Royal College of Surgeons, Howkins[7] stressed once again the sites in the pelvis where the ureter is most prone to injury. These are:

1. Where the ureter is crossed by ovarian vessels contained in the ovariopelvic fold of peritoneum — a healthy distance away in the normal individual, but often dangerously close following inflammation, or where adnexal tumours are present.

2. As the ureter clings to the peritoneum through most of its pelvic course, inflammatory or malignant disease causing infiltration will make it susceptible to injury.

3. In its medial course in the base of the broad ligament, to reach the ureteric tunnel in Mackenrodt's ligament where it passes beneath the uterine vessels, 1·5 - 2 cm. from the cervix, the ureter lies in greatest peril during hysterectomies. This is also the common site of strangulation by parametric malignant infiltration.

4. After tunnelling the lateral part of Mackenrodt's ligament, the ureter traverses the anterior part of the same ligament — the so-called pubocervical ligament, at which point it is in special danger in all radical operations for carcinoma of the cervix. According to Grant[8] it lies closer to the cervix here on the one side than on the other. (Usually closer on the left.)

5. Finally it is stressed that over-enthusiastic needle and suture reperitonealization of raw areas may easily produce piercing, occlusion, or kinking of the ureter, far more commonly than one normally imagines.

Duffy[9] stresses, and shows radiologically, the very gross downward displacement of the ureters that occurs in severe genital prolapse, particularly in procidentia. This makes them especially vulnerable in operations designed to cure these prolapses or cystoceles, and in vaginal hysterectomies.

Pathology

There is fairly general agreement that injury occurs most commonly during the following operations — in order of frequency:

(1) Radical abdominal hysterectomy, (2) total abdominal hysterectomy, (3) subtotal hysterectomy, (4) vaginal hysterectomy, (5) salpingo-oophorectomy, and (6) intraligamentous tumours.

However, there appears to be no gynaecological procedure, apart from the humble dilatation and curettage, that has not been complicated at one time or another by injury to one or other ureter, not excluding anterior colporrhaphies and Fothergill procedures. Nor is the work of the obstetrician entirely free from risk in this regard, for at least 5 cases have followed the application of forceps, and at least 3 cases, Caesarean section. Nor is ureteric injury the prerogative of the inexperienced or poor surgeon. There is unanimous agreement that injury to the ureter may occur in the hands of both the most highly skilled and adept surgeon, and his less able colleagues — perhaps even more so in the former group because of the greater tendency to carry out the many more radical procedures.

Types of Injury

The following list includes all possible types of injury: (*a*) Partial or complete ligation; (*b*) crushing of the wall, complete or incomplete; (*c*) complete division; (*d*)

resection, accidental or deliberate, as in carcinoma surgery; (*e*) angulation or kinking; and (*f*) necrosis due to interference with blood supply.

The incidence of each particular type of injury is very difficult to assess, but in Badenoch's[10] series of 39 cases, more than 50% were found at operation to be due to suture or ligature. With such a high incidence of major injury it seems both illogical and dangerous to base therapy, or withhold therapy, on the premise that one or other less serious type of injury may be the cause of the obstruction.

Symptoms

Symptoms are extremely variable. Pain in the lower abdomen and the affected flank, accompanied by fever in the infected case, with or without some degree of ileus, can all be anticipated, particularly in those cases that are incompletely obstructed. Most authors agree that there may be no symptoms whatsoever that direct attention to the kidney, because loin pain is not a feature. In fact, Patton[11] states quite categorically that 50% of all cases of proved ureteric injury have no renal pain at any time. However, where pain is present, the relief following fistula formation is dramatic and complete. Anuria following operation, in the absence of any prerenal cause, is the obvious symptom of bilateral ureteric involvement. On the other hand oliguria, while tending to raise suspicion of unilateral injury, is much more difficult to assess and therefore of less consequence. Fistula, which is incontrovertible evidence of injury in the urinary tract, may occur at any time up to the 3rd or 4th week. In Badenoch's[10] series 20% occurred in the 1st week, due, he thinks, to division or partial inclusion in a suture; 60% occurred in the 2nd week, due to ligation and subsequent sloughing; and the final 20%, which occurred in the 3rd week, are according to him, due to ischaemia. A healthy suspicion on the part of all gynaecologists should make daily renal palpation after all pelvic operations a normal, necessary, never-to-be-forgotten routine; any tenderness of one or other kidney should make intravenous pyelography imperative; unexplained pyrexia, abdominal distension, or ileus, should create a suspicion of ureteric obstruction, complete or incomplete.

Diagnosis

Apart from the symptoms and signs discussed, the only certain method of diagnosis is by pyelography. In St. Martin's[5] series the injuries were asymptomatic in over 30% of the cases, the diagnosis being made solely by urography. (This is the series, mentioned before, where routine pre- and postoperative urographic studies were carried out.) Usually the ureteric catheter is held up at the site of injury. In some few cases of partial tear or partial injury, the catheter may rarely be manoeuvred past this site and, where this has been achieved, it can be left indwelling as a therapeutic measure. The site of injury is usually up to 8 cm. from the ureteric orifice, being as a rule at a higher level after abdominal surgery than after vaginal surgery.

TREATMENT

Prevention

A. Pre-operative

Wherever there is a possibility of ureteric damage in major pelvic surgery, preliminary intravenous pyelography should be carried out. In all cases of carcinoma of the cervix this should be mandatory, not only because of possible damage at operation, but also to provide information concerning early ureteric involvement due to spread of the disease. Where intravenous pyelography reveals distortion or obstruction, it seems almost axiomatic for the cautious surgeon to have the ureters catheterized during the course of the operation. This is, in fact, the practice of many gynaecologists, among them Conger and his associates[4] of Philadelphia, who remove these catheters before the abdomen is closed. In this way they check carefully for any resistance indicative of ureteric occlusion. Landsteiner,[12] Te Linde,[13] and Thompson and Counsellor[14] of the Mayo Clinic all advise indwelling ureteric catheters as a precautionary procedure in *difficult* gynaecological surgery.

B. At Operation

1. If there is a possibility of damage to the ureter, this structure should be isolated at the bifurcation of the iliac vessels, followed down carefully, and retracted out of harm's way, as a definitive preliminary procedure. Graham and Goligher[15] do this as a routine in operations for excision of the rectum.

2. As severe and at times almost uncontrollable haemorrhage may occur in the pelvis, both arterial and venous, gynaecologists might be well advised to adopt the procedure normally used by urologists in dealing with the slipped renal pedicle, viz. packing the cavity with large packs for 20 minutes by the clock, and then clamping and ligating only the offending vessels.

3. Very careful reperitonealization so as to avoid puncture, occlusion by the stitch, or angulation of the ureter, should be carried out; with a catheter *in situ*, the ureter can be more easily avoided. Howkins[7] feels that interrupted tacks, producing closure without tension, are far preferable in this respect to a continuous suture.

Injuries Recognized at the Time of Operation

The principles underlying treatment are: (1) Conservation of renal tissue, (2) restoration of normal anatomy as far as possible, and (3) the performance of the simplest procedure that suits the particular case.

(*a*) Crushing injuries noted at the time are treated by immediate removal of the clamp. Where no obvious injury appears to have been caused to the ureter, a ureteric catheter should be inserted at the end of the operation and should remain indwelling for 8 - 10 days.

(*b*) Where definite injury to the ureteric wall is noted, it is preferable to excise the injured area and to do an immediate anastomosis, if possible, preferably using the technique of Hamm and Weinberg;[16] failing this, the ureter should be re-implanted into the bladder. Extraperitoneal drainage is mandatory in either case. Most of the injuries to the pelvic ureter can be dealt with in this way, but where there is marked loss of ureter, reconstruction by using a bladder flap after the method of Boari and Ockerblad may have to be substituted. Although some surgeons recommend transplantation to the unprepared sigmoid or caecum, this method is to be deplored because of the obvious danger of ascending pyelonephritis; temporary cutaneous ureterostomy is to be preferred.

Ligation of the cut ureter, unless a very large segment has been removed, making any other method impossible, cannot be too strongly condemned, as it was by John Bland-Sutton,[17] who stated: 'Kidney tissue is very precious. Many persons get on very well in the world with very little brains, and some with none at all, but no one can live without a certain amount of kidney tissue'. Howkins contends that ligation of a cut ureter is the resort of the poor surgeon, and that it is a dishonest and dangerous shortcut out of a difficult situation. Moreover, according to Graham and Goligher,[15] 5 out of 7 cases with tied ureters required nephrectomy eventually and others, still more unlucky, have reached the postmortem room, showing urinary extravasation and peritonitis as the cause of death.

Injuries Recognized After Operation

(*a*) *Bilateral.* There is much difference of opinion regarding the management of patients in whom injuries are recognized after operation, both because the patient is, on occasion, very ill and also because of the fact that the exact nature of the obstruction, which is itself so important, can never be ascertained with any degree of certainty until exploration is undertaken. Early diagnosis is most essential. If no urine is present in the bladder after 24 hours, immediate investigation must be undertaken. Cystoscopy and the passage of ureteric catheters will usually define the sites of obstruction.

After operations for repair of the pelvic floor, the removal of *all* the sutures may be all that is required to reverse the anuric condition. After other procedures, providing the patient's general condition is good (and it usually is good 24 - 48 hours postoperatively) immediate bilateral deligation is recommended by Reisman and his associates,[18] Badenoch,[10] and Moore.[19] Other surgeons prefer bilateral nephrostomies as a preliminary procedure, with surgery to the lower ureter at a much later date — up to 18 months later. The most impressive report in the literature is that of Reisman and his associates[18] who detail 7 consecutive cases of unilaterally ligated ureters that were successfully deligated between the 5th and 33rd postoperative days, each as a primary procedure. They emphasize the necessity for cooperation between surgeon and urologist (or 2 urologists working together, one abdominally and the other cystoscopically) with ureteric catheters bringing into relief the site of obstruction. The approach is probably better extraperitoneally, through a clean field, rather than through the original approach. Badenoch[10] favours this extraperitoneal approach. Deligation and splinting for 8 - 10 days seems, in the series mentioned, to have produced, in one operation, uniformly excellent results. While bilateral nephrostomy cannot possibly be justified as a life-saving procedure, unilateral nephrostomy must be accepted, in the very ill patient, as a necessary, but rather inferior, method of treatment. However, according to Reisman[18] none of the patients appears to be so ill, even by the 6th postoperative day, so that the procedure of choice should be immediate deligation, so producing a normal urinary tract in one, and not multiple, operations. The plea that a small number of obstructed ureters may re-open after preliminary nephrostomy merely encourages delay and multiple procedures that, in the light of Reisman's[18] report and Badenoch's[10] operative findings, would now be hard to justify.

(*b*) *Unilateral.* There is scant justification for preliminary nephrostomy in those cases where a normally functioning contralateral kidney is present; immediate deligation and splinting being the procedure of choice here. If a large section of the ureter is strictured, excision and re-anastomosis or re-implantation into the bladder is advocated. At this juncture, a word about the technique of re-implantation would not be out of place. All sorts of operative procedures have been devised from time to time in order to prevent reflux up the ureter after ureteroneocystostomy. While Patton[20] makes use of the original ureteric orifice, others produce elaborate oblique courses through the muscle coats, nipple anastomoses of different types, and various similar procedures. While Thompson and Counsellor[14] have stated that re-implantation into the dome fails because of regurgitation, Riches[21] believes that ureteric reflux is no more common using this method than after more ingenious procedures; nor is the method particularly liable to lead to stricture formation at the anastomosis. Certainly there has never been sufficient ureter to carry out Patton's[20] procedure in my cases, in all of which anastomosis to the nearest adjacent portion of the bladder, after fishmouthing the ureteric orifice in the manner of Marion, has been performed. A plastic ureteric splint is left indwelling for 10 days, the anastomosis at times being under very marked tension. Neither upper urinary tract dilatation nor pyelonephritis has so far resulted following the use of this simple method.

Ureterovaginal Fistulae

As many gynaecologists feel that a course of watchful inactivity is justified, believing, as Hinman[22] did, that 50% of ureterovaginal fistulae heal spontaneously, this condition is one that should be investigated and treated by urologists as soon as it is even suspected. This is all the more so, since the investigation and plastic surgery that follows, are primarily urological procedures. The few ureterovaginal fistulae that do 'heal' spontaneously, heal by stricture formation and autonephrectomy. In many kidney function is irretrievably lost, but even where some function persists, the affected upper urinary tract is certainly not normal, particularly where the fistula has taken many weeks to heal, with attendant bouts of pyrexia and loin pain on the affected side. The urologist, interested as he is in primarily preserving renal function, and in the re-establishment of normal anatomy, will adopt a more active approach to the urinary leak — to the greater comfort of the patient and a reasonable expectancy of normal renal function. Although Thompson and Counsellor[14] report that in as many as 8 out of 34 cases they could find no dilatation proximal to the site of leakage, other investigators have found not only dilatation but also gross pyelonephritis. There is no doubt, as may be seen from the pyelograms here reproduced, that dilatation is extremely rapid, and although kidneys obstructed by ligature for up to 33 and 81 days have recovered function after re-implantation of the ureter into the bladder, it would be foolish to anticipate as a routine such unexpected and happy results. My plea

is for treatment to be undertaken as soon as the condition is diagnosed, and with this Millin[23] concurs. Howkins[2] advises delay of up to 6 months, during which period the unfortunate patient is either confined to her wet bed, with a packed vagina, connected to a low suction drainage apparatus, or gets around most uncomfortably in pads and waterproof panties — a form of management that merits the strongest condemnation. While ideal treat-

Fig. 1. M.P. Pre-operative (1 month post-hysterectomy); showing hydronephrosis and hydro-ureter. Site of obstruction obscure.
Fig. 2. M.P. Postoperative; showing restoration of function and anatomy.

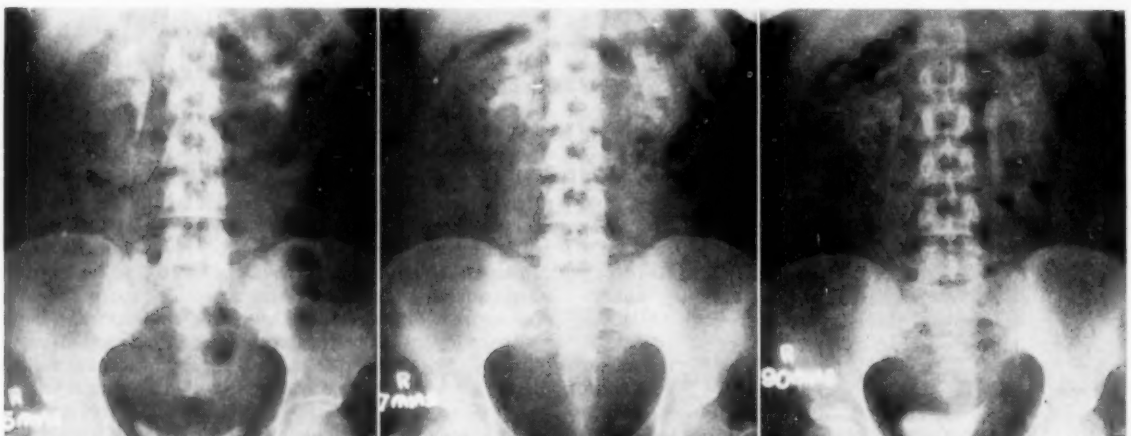

Fig. 3. J.S. Pre-operative; showing gross dilatation on the left side and puerperal dilatation on the right side.
Fig. 4. J.S. Postoperative (1 month); showing rapid return of function.
Fig. 5. J.S. Postoperative (1 month); showing diminished dilatation with marked tenting of the bladder at anastomosis. (This disappears gradually.)

ment should consist of re-anastomosis of the ureter. Millin[23] and Badenoch[10] state that this concept is largely theoretical and usually impracticable, and that the best that can be hoped for is a successful re-implantation into the bladder. This is my impression too, and probably that of all urologists who tackle these cases early, since the pelvic mass in which the stricture and fistulous track lie is formidable and better left well alone.

The following case histories of 3 ureterovaginal fistulae illustrate some of the points mentioned — the first 2 cases are routine, simple and straightforward; the third presents a somewhat more complex picture.

Case 1

M.P., aged 37 years, had a right ovarian cystectomy 10 years previously. For the last 2 years she had complained of pain in her right groin with irregularity of her periods. A subtotal hysterectomy and right oophorectomy, for chocolate cyst of the ovary, were carried out 1 month previously without any apparent untoward incident at operation.

About 10 days after the operation she started complaining of pain in her lower abdomen which persisted for 2 days, after which urine started leaking vaginally. With the leakage of urine the lower abdominal pain disappeared. Intravenous pyelography (1 month after hysterectomy) showed normal function and anatomy of the upper urinary tract on the left side. On the right side there was grossly delayed function with hydronephrosis and hydroureter. The level of the obstruction could not be made out (Fig. 1). Cystoscopy and retrograde pyelography showed no efflux on the right side with complete blockage of the right ureter at 6 cm. Attempted retrograde pyelography resulted in reflux of dye back into the bladder.

Operative findings (5 weeks after hysterectomy): The right ureter was found to be grossly dilated, disappearing into a mass of scar tissue at the level of the iliac bifurcation. The ureter was divided just above the bifurcation and re-implanted into the bladder, at the most convenient site on the dome and under slight tension, over a No. 3 polythene tube, using the technique of Marion.

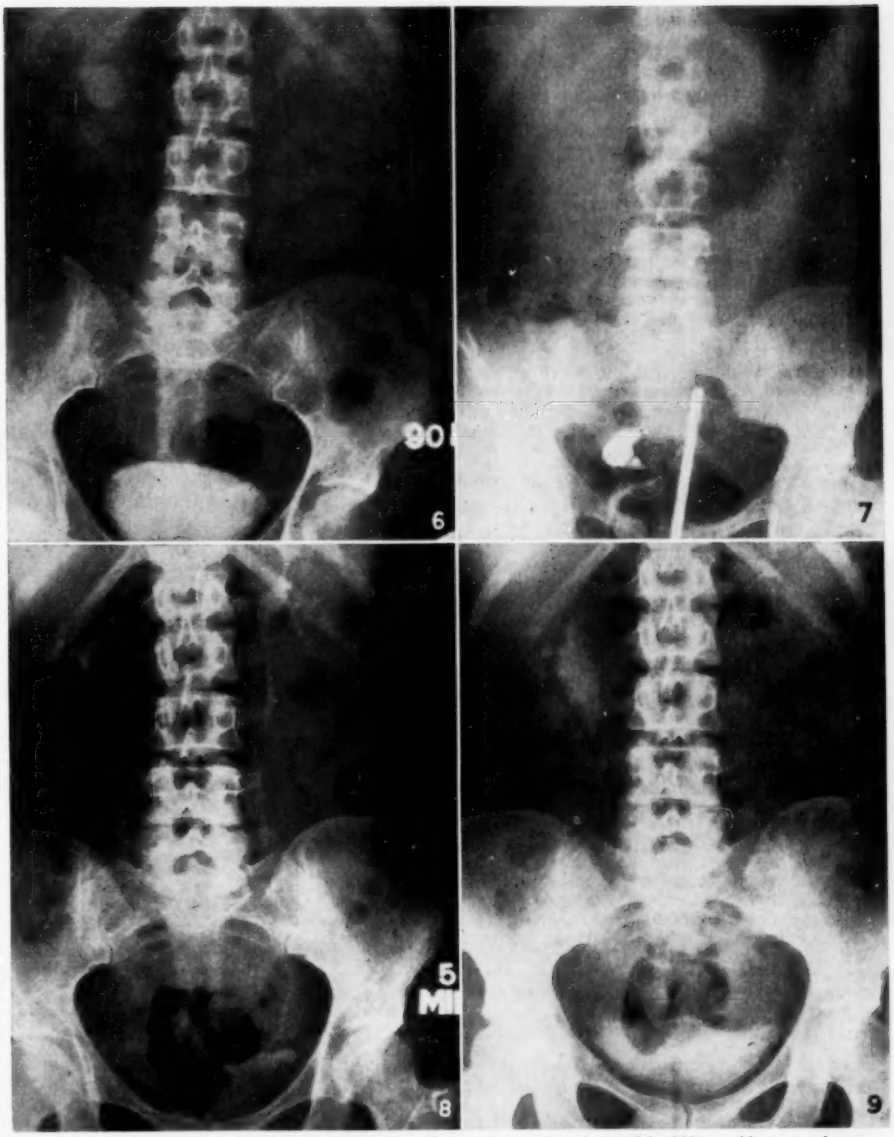

Fig. 6. G.S. Pre-operative; showing gross dilatation of the minor calyx of the right kidney with severe depression of function.

Fig. 7. G.S. Retrograde pyelogram showing dye extravasation through a small cavity into the vagina.

Fig. 8. G.S. Postoperative restoration of good function.

Fig. 9. G.S. Postoperative restoration of near-normal anatomy. Note the congenitally dilated atonic kidney pelvis.

Postoperative films show normal function and anatomy on the right side (Fig. 2).

Case 2

J.S., aged 26 years, had 3 Caesarean sections over the last 6 years for toxaemia of pregnancy. The last one was performed 3 weeks previously and, according to her gynaecologist, was a very difficult procedure with a bleeding vessel on the left side of the pelvis constantly slipping away. This bleeding vessel was finally underrun to control the haemorrhage.

Her postoperative course was grossly pyrexial despite anti-

biotics, and she complained of intermittent pain in the left loin from the 4th day onwards. Vaginal urinary leakage commenced about 10 days postoperatively, from which time the pyrexia had settled.

Physical examination revealed a tender, slightly enlarged, left kidney with tenderness over the course of the left ureter down to the level of the anterior superior iliac spine.

Intravenous pyelography (3 weeks after Caesarean section) showed normal function on the right side and grossly delayed excretion on the left. The right kidney and ureter showed normal puerperal dilatation, and the left kidney and ureter showed evidence of marked obstruction, the exact site being obscure (Fig. 3).

Left retrograde pyelography showed obstruction at 7 cm. with extravasation of dye outside the ureter. Ureteroneocystostomy was performed 5 weeks after the birth of her child. The left ureter was found to be dilated down to the brim of the true pelvis where it disappeared into a mass of tissue extending down to the broad ligament. This ureter was divided above the mass and re-implanted into the dome of the bladder, over a polythene splint, under very considerable tension. Despite this tension, which is visible in the postoperative pyelograms shown, there was rapid return of function (Fig. 4) and marked diminution in dilatation of the upper urinary tract. 1 month after re-implantation (Fig. 5).

Case 3

G.S., aged 40 years, complained of urinary incontinence for 2 weeks and gave a complicated history.

She was last well about 4 months previously when a hysterectomy was performed for persistent menorrhagia. Her gynaecologist reported that a fibroid uterus and a right cystic ovary were removed and that there was more than average oozing throughout the operation, particularly during wound closure. Her convalescence was quite uneventful and she was discharged after 11 days. Two months later she complained of severe lower abdominal pain of rather sudden onset without any other sign than abdominal distension. Diagnosed as a case of subacute intestinal obstruction, she was re-operated on for adhesions, which were divided, the surgeon finding that the site of right oophorectomy was adherent to the rectal wall. Following this operation she was well for about 5 weeks, when she experienced severe burning high up in the vagina for a week. This burning was relieved when urinary leakage commenced *per vaginam;* she had been incontinent since. Intravenous pyelography (4 months after her hysterectomy) showed a normal left upper urinary tract, both functionally and anatomically. On the right side there was poor concentration though the dye appeared early, with very gross hydrocalycosis. Neither pelvis nor ureter showed throughout the series (Fig 6).

Retrograde pyelography showed an obstructed right ureter 7 cm. from the ureteric orifice. A small cavity communicating with ureter and vagina was outlined (Fig. 7). Speculum examination showed the fistulous opening in the vaginal vault on the right side.

Right ureteroneocystostomy was performed 4½ months after hysterectomy. At the operation a thickened, grossly dilated, ureter was seen to disappear into a mass of tissue in the neighbourhood of the superior vesical pedicle. The ureter was cut across above the mass and re-implanted into the side of the bladder with minimal tension.

Postoperative films (5 months after the operation) showed normal function (Fig. 8) with a congenitally large atonic pelvis. It is probable that renal function was conserved so long because of this atonic pelvis relieving the calyces of maximal back-pressure after obstruction had commenced (Fig. 9).

SUMMARY

1. The vulnerability of the pelvic ureter in gynaecological surgery is stressed.

2. The aetiology, pathology, diagnosis, and management of ureteric injuries at all stages, are discussed.

3. A plea is made for greater awareness which will lead to earlier recognition of injury, earlier definitive urological care, and restoration of renal function.

4. Three cases of ureterovaginal fistulae are specifically quoted to illustrate some of the above points.

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FORTHCOMING INTERNATIONAL MEDICAL CONFERENCES

The Third International Congress of Dietetics will be held in London on 10 - 14 July 1961. Lord Boyd Orr is the Hon. President and the Congress is organized by The International Committee of Dietitians and The British Dietetic Association. Two international congresses have already been held, one in Amsterdam in 1952 and one in Rome in 1956. The 1961 Congress will be at Church House, Westminster, London, S.W.1. Final announcements and forms for registration and accommodation will be circulated towards the end of 1960. The Congress languages will be English, French and German, with publication of proceedings in English or French. There will be simultaneous interpretation at the main sessions.

Frames Tours Ltd. are the official travel agents for the Congress and will make hotel reservations. At the moment the price per night of a hotel room with breakfast ranges from 25s. to 50s. Some accommodation at lower prices will be available at university halls of residence. Frames Tours Ltd. will arrange post-congress tours of the UK, if required.

The theme of the Congress is 'Tradition, science and practice in dietetics'. The opening symposium on the history of human diet will be introduced by Dr. N. C. Wright, Deputy Director-General of the Food and Agriculture Organization of the United Nations and by Mr. F. Le Gros Clark (UK). Under the chairmanship of Prof. E. Neige Todhunter (USA), this discussion will continue with contributions from 5 parts of the world. A symposium on the education and occupations of dietitians will be under the chairmanship of Miss J. A. S. Ritchie (FAO), with speakers from 7 countries. Other discussion groups during the Congress include 'Diet and ischaemic heart disease' (Chairman Prof. John Yudkin, UK); 'Therapeutic dietetics' (Chairman Prof. Sir Derrick Dunlop, UK); 'Nutrition of mother and child' (Chairman Prof. B. S. Platt, UK); and 'Feeding old people'.

Enquiries should be addressed to the Office of the Secretariat, 251 Brompton Road, London, S.W. 3, England.

South African Medical Journal : Suid-Afrikaanse Tydskrif vir Geneeskunde

EDITORIAL : VAN DIE REDAKSIE

URETERIC INJURIES IN GYNAECOLOGY

It behoves all surgeons, throughout their careers, repeatedly to examine, re-assess and re-appraise their methods of investigation and treatment in the light of continued surgical advance. All too often outmoded procedures and forms of therapy are perpetuated long after the foundations for their continued use have been shown to be faulty, insecure, and unstable — to the detriment of the patient. The surgery of the injured ureter is a case in point. In the past diagnosis was often incomplete, delayed, or completely missed. As a result treatment was delayed with increased morbidity and often gross destruction of kidney tissue. The more modern approach stresses the avoidance of injury, outlines means of early and complete diagnosis and methods of direct surgical attack.

Perhaps the most important single factor responsible for the changed surgical approach has been the increased use of intravenous pyelography, both pre- and postoperatively, as a diagnostic aid. Very few people are too ill, even 36 - 48 hours after operation, for intravenous pyelography, which is a procedure of minimal danger causing only slight inconvenience, at worst. It is a procedure requested all too infrequently by gynaecologists.

It has become patently obvious, using this method of investigation, that the ureter, in its course through the female pelvis, is injured surgically far more often than we have been led to believe in the past. Abundant articles in the literature confirm this. It is further an accepted fact that a large proportion of these injuries are first discovered, either in the postmortem room or in the urological department, at some later date, when they present as gross hydronephroses or non-functioning kidneys. The symptoms of ureteric injury are notoriously silent in as high as fifty per cent of cases, even after complete ureteric ligation. The occasional backache in the postoperative period is too often accepted as of little import.

It is thus timely that Mr. I. Jacobson, in his article in the present issue of the *Journal*, should shed a little more light on the incidence of this much-neglected injury and its management; all the more important because of the particular attention paid in this article to controversial methods of management. It is also interesting to note that Mr. A. Dickson Wright, of London, during his recent visit to this country, chose, as the subject of his Margaret Orford Memorial Fund Lecture 1960: 'Urinary complications of gynaecological surgery'. The fact that both these authors, independently of one another, draw attention to this problem at this stage, is a significant reflection of the mood of present trends in this connection.

A full appreciation of the incidence, sites of particular danger, methods of avoidance of injury, and early recognition both at operation and immediately after operation, in addition to the ability to deal with such injuries on the spot, should all be part of the working knowledge of the well-trained gynaecologist. Where he feels unable to handle the complication, early recognition of the condition will mean earlier definitive urological surgery.

Ligation of the cut ureter is still carried out today far more frequently than is necessary — almost certainly because of a lack of full appreciation of alternative methods of management. Bland-Sutton condemned this treatment at the turn of the century as the poor surgery of a destitute surgeon. That condemnation is as true today as it ever was, nor lightened in any way by the possible (but, usually, unproved) presence of a normal kidney on the contralateral side. The ureteric injury, unavoidable as it sometimes may be, appears to be sufficient additional burden for the patient without the all-too-hasty, immediate, enforced loss of half her renal tissue. Though at times the kidney may have to be sacrificed, that should remain the last, and not the first, line of treatment.

The most satisfactory treatment for postoperative bilateral ureteric obstruction after pelvic surgery is also somewhat contentious. The old method of treatment consisting of bilateral nephrostomies with plastic procedures to the lower ureters to follow, entails at least three operations as a rule, with a delay of up to 6 - 18 months between the last two. The more modern conception of immediate deligation of both ureters, performed extraperitoneally, as quoted in Jacobson's article, gives immediate restoration of function as soon as possible — in one procedure. The patients do not seem to be too ill for this procedure at any time in the immediate postoperative period, until uraemia supervenes. This approach has recently been advocated on both sides of the Atlantic and, according to the results of Reisman *et al.*[1] appears to be well worthy of extended trial by urologists.

Without doubt the most unpleasant complication from the patient's point of view is a ureterovaginal fistula. It has long been the teaching that fifty per cent of these cases (Hinman[2]) heal without surgical interference. As a result gynaecologists will usually adopt an attitude of 'wait and see' for as long as the patient and her relatives will let him — preferably for up to six months. If the fistula should heal during this time, it is almost certainly as a direct result of complete loss of renal function on the affected side and therefore no valid reason for self-congratulation on the part of the surgeon. If healing does not take place, recurrent attacks of pyelonephritis continue to sap renal reserve with increasing destruction of renal tissue, making eventual nephrectomy a far too common sequel after months of extreme discomfort and misery for the patient.

Mr. Jacobson, in his article, makes a plea in these cases, where the diagnosis is patently obvious as a rule, for immediate urological investigation. The surgical management of this complication is essentially within the compass of the modern urologist who is quite prepared to re-implant the affected ureter into the bladder as soon as possible after diagnosis has been confirmed radiologically. The results of these re-implantations, judging by the cases reported in Jacobson's article, appear to be uniformly satisfactory. The patient is rapidly relieved of the condition which makes her, temporarily at least, a social outcast and, even more important than this consideration, renal functioning tissue is conserved and preserved. Only by treatment along these lines can the tragedies of the injured pelvic ureter be averted.

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PRIMÊRE PIGMENTASIE

Primêre of direkte pigmentasie moet onderskei word van sonbrand-pigmentasie wat gewoonlik enkele dae na sonblootstelling plaasvind en wat gewoonlik volg op 'n sonbrand-eriteem. In die geval van primêre pigmentasie vind die verdonkering reeds tydens die bestraling plaas. Dit is gewoonlik binne tien minute na blootstelling aan direkte sonlig baie duidelik waarneembaar veral by die gekleurde rasse. Die verskynsel is die eerste keer in 1938 deur Hauser[1] en, onafhanklik van hom, in 1938 deur Henschke en Schulze[2] waargeneem by bestraling met ultraviolet ligbronne.

Aanvanklik is primêre pigmentasie beskou as 'n taamlik ongewone reaksie wat net by sommige mense voorkom. Kooij en Scott[3] kon egter aantoon dat dit 'n algemene verskynsel is by gekleurde rasse en dat dit selfs by Blankes maklik op te wek is indien daar reeds pigment aanwesig is. In hierdie uitgawe van die *Tydskrif* word 'n oorsig van werk wat reeds op hierdie gebied gedoen is, gegee en verdere eksperimente word gerapporteer.

Dit is verbasend dat in die verlede so weinig aandag aan hierdie verskynsel, wat so maklik by gekleurde rasse op te wek is, gegee is. In hulle eerste publikasie oor hierdie onderwerp het Kooij en Scott[3] met die hulp van filters die aktiewe spektrum vir hierdie verskynsel op ongeveer 300 - 460 mμ vasgestel. Dit verskil dus van die kortgolwige ultraviolet-spektrum van die son wat tussen 290 - 320 mμ lê en wat vir die sonbrand-pigmentasie verantwoordelik is.

In die artikel waarna ons verwys word verder gerapporteer oor die invloed van verskillende stowwe op hierdie verskynsel. Geeneen van die veertien stowwe het by plaaslike aanwending enige invloed op die verskynsel gehad nie. Een stof egter, nl. metoksalen, 'n furokumarien, het 'n duidelike invloed op die sonbrand-reaksie vertoon. Met die hulp van verskillende filters kon aangetoon word dat hierdie stof verantwoordelik is vir verskuiwing van die sonbrand-verwekkende strale na 'n langer golflengte. Hierdie bevinding, naamlik die veranderde gevoeligheid vir sonstrale, mag nuwe lig werp op die uiters belangrike vraagstuk van siektes wat ontstaan op die basis van oorgevoeligheid vir sonstrale — 'n groep aandoeninge wat 'n belangrike rol by die huidpatologie van ons sonryke land speel. Die skrywers spekuleer oor die moontlikheid van abnormale stowwe (metaboliete) wat onder die invloed van sonlig ontstaan en verantwoordelik is vir die abnormale reaksie.

Met hierdie werk wat in Suid-Afrika gedoen word, word 'n verdere bydrae gelewer tot die ingewikkelde meganisme van pigmentvorming. Of ons hier net met 'n eenvoudige pigmentasie van 'n ligtere na 'n donkere soort melanien te doen het, kan alleen deur verdere eksperimente aan die lig gebring word. Ook die verband met die alreeds lankbekende postmortale verdonkering (‚pigment darkening') moet nog aangetoon word. In 'n volgende artikel word hierdie verskynsel behandel en word daar oor eksperimente met uitgesnyde stukkies huid gerapporteer.

Suid-Afrika met sy oorvloed van sonlig en uitstekende materiaal bied 'n gulde geleentheid vir studies op hierdie gebied. Die Fotobiologiese Navorsingsgroep van die W.N.N.R., onder wie se beskerming hierdie navorsing plaasvind, behoort 'n belangrike bydrae tot die huidpatologie in ons land te lewer.

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OBSERVATIONS ON PRIMARY AND POSTMORTEM PIGMENTATION BY SUNLIGHT*

I. PRIMARY PIGMENTATION. ACTION SPECTRUM OF METHOXSALEN

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Primary pigmentation, which was discovered independently by Hauser[1] in 1938 and Henschke and Schulze[2] in 1938, is not a well-known phenomenon. It was also studied in detail in 1939 by Miescher and Minder.[3] Primary pigmentation occurs almost immediately after, or even during, exposure to sunlight, whereas the well-known secondary pigmentation (suntan) only occurs a few days after exposure.

The wavelengths responsible for primary pigmentation lie roughly between 300 and 460 mμ, while those for secondary pigmentation lie between 230 and 320 mμ. No ultraviolet rays from the sun shorter than 290 mμ reach the surface of the earth. Primary pigmentation can easily be produced in Coloured races after 10 to 30 minutes of exposure to sunlight.

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It is assumed that no new pigment is formed in primary pigmentation but that a simple oxidation of paler melanin to darker melanin takes place. The process is reversible; fading usually starts 30 to 60 minutes after exposure and disappears completely in 1 or more days.

The general occurrence of this phenomenon in coloured races was discovered accidentally by Kooij and Scott[4] in 1953 while experimenting with sunlight in a Coloured patient suffering from chronic porphyria.

More than 100 Bantu and Coloured subjects studied by them showed this phenomenon without a single exception. Although it could not be evoked in albino Bantu subjects it could be demonstrated in about one-third of White subjects exposed to sunlight, as it is found only when pigment is already present. Direct erythema often occurred simultaneously with the pigmentation, usually when filters transmitting wavelengths above 460 mμ were used.

With the aid of Ilford filters it was possible to show that the wavelengths between 300 and 460 mμ were responsible for the primary pigmentation. The slight increase of pigment in the basal layer described by Hamperl, Henschke and Schulze[5] and Miescher and Minder[3] was confirmed by frozen-section examinations made immediately after exposure.

Continued studies were done at Cape Town by one of us (R.K.) and at Bloemfontein by the other (F.P.S.) to establish the influence of different locally-applied substances on the phenomenon, and to investigate the occurrence of primary pigmentation after death and after excision of skin (see part II, to be published later).

MATERIAL AND METHODS

The experiments were carried out on a number of non-White subjects. Each substance tested was applied twice on the back of every subject — 2 hours before exposure to sunlight and also immediately before this exposure. Those substances applied 2 hours before were removed before exposure, but those applied immediately before the exposure were left on during the period of exposure.

The following substances were tested: 5% tannic acid in 95% ethyl alcohol, 5% chlorpromazine in 95% ethyl alcohol, 5% sulfanilamide ointment, anthisan ointment, 15% hydrogen peroxide in eucerin anhydricum, 10% ascorbic acid in eucerin anhydricum, 1% testosterone phenyl propionate in ethyl alcohol, 20% benoquin ointment, 2½% hydrocortisone ointment, ¼% oestrogens (premarin) in aqueous solution, ½% aqueous solution of copper sulphate, 1% aqueous solution of sodium fluoride, methoxsalen (8-methoxypsoralen) $^1/_{10,000}$, $^1/_{3,000}$, and $^1/_{1,000}$ in 95% ethyl alcohol, and 1% haematoporphyrin in ethyl alcohol.

The areas were exposed to sunlight for 20-minute periods between the hours of 2 p.m. and 3 p.m. during the months November to March. The results were read immediately after exposure and the areas were kept under close observation during the first hours and also for several days thereafter. The degree of erythema and pigmentation was estimated with the naked eye and marked +, ++, and +++. The exposed areas were covered with glass slides so as to cut out wavelengths below 320 mμ.

In some instances Schott filters were used. For details about the transmission (absorption spectra) of these filters see Table I.

TABLE I. THE ABSORPTION SPECTRA OF THE SCHOTT FILTERS USED*

Wavelengths in mμ	Filters							
	UG3	UG4T	UG5	UG11	WG1	GG3A	GG14	ARGIA
203	—	—	—	—	—	—	—	—
229	—	—	0·27	—	—	—	—	—
254	—	—	0·73	—	—	—	—	—
281	—	—	0·94	0·67	—	—	—	—
302	—	0·02	0·98	0·88	—	0·03	—	—
312	0·04	0·12	0·99	0·93	—	0·05	—	—
334	0·56	0·54	0·98	0·98	—	0·10	—	—
366	0·91	0·77	0·90	0·93	0·48	0·17	—	—
405	0·85	0·09	0·38	—	0·98	0·22	—	—
436	0·49	—	0·10	—	0·99	0·84	—	—
480	0·17	—	0·02	—	1·00	0·99	—	—
509	0·12	—	—	—	1·00	1·00	0·96	—
546	0·14	—	—	—	1·00	1·00	0·99	—
578	0·21	—	—	—	1·00	1·00	0·99	—
644	0·38	—	0·02	—	1·00	1·00	0·99	0·96
700	0·52	0·20	0·71	0·35	1·00	1·00	0·99	1·00
775	0·68	0·53	0·58	0·05	1·00	1·00	1·00	1·00
850	0·84	0·35	0·51	—	1·00	1·00	1·00	1·00
950	0·95	0·34	0·52	0·03	1·00	1·00	1·00	1·00
1050	0·98	0·29	0·53	0·08	1·00	1·00	1·00	1·00

All filters used were 1 mm. in thickness.

* Adapted from tables supplied by Schott and Gen., Jena Glassworks, Main., Germany.

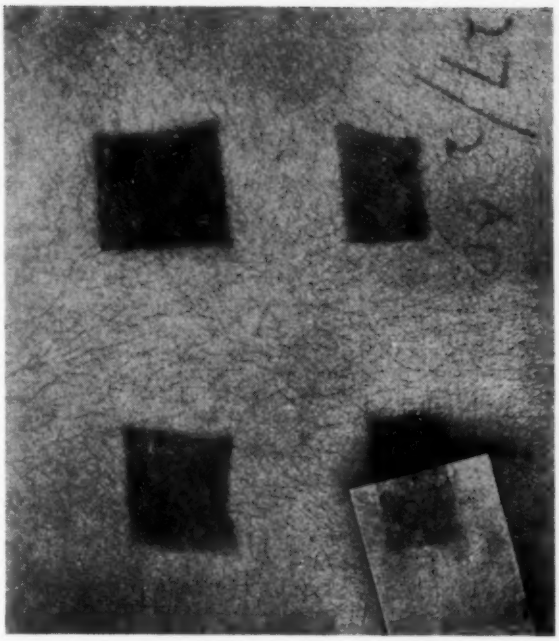

Fig. 1. Delayed erythema and pigmentation of areas treated with methoxsalen $^1/_{10,000}$ - $^1/_{1,000}$ following exposure for 20 minutes to sunlight which passed through glass. Photograph taken 3 days after exposure. Primary pigmentation had disappeared after 1 day.

RESULTS

The above substances did not exert any noticeable influence on primary pigmentation as compared with the controls. The duration and the degree of pigmentation was the same in both.

Primary pigmentation occurred in all the non-White subjects tested. It usually faded considerably after 30 minutes and sometimes it was hardly visible the next day. It could however often be observed several days after exposure. The duration of the primary pigmentation is probably due to the intensity of the irradiation and the ability of the individual to tan.

An interesting fact was observed in the exposed areas treated with methoxsalen. These areas showed a faint erythema on the day following the experimental exposure to sunlight. The primary pigmentation had disappeared by this time. The erythema gradually became stronger and was marked the next day, when slight pigmentation became

visible; this increased in intensity over the following days.

This delayed erythema followed by pigmentation also appeared when the methoxsalen-treated areas were covered with a glass slide, which cut out the 'sunburn rays' below 320 mμ (Fig. 1). No such reactions occurred in the areas treated with the other substances. With the aid of the absorption filters UG3, UG4T, WG1, and GG3A an attempt was made to determine the active wavelengths of this phenomenon (for details about the transmissions of these filters see Table I). The delayed erythema and pigmentation could only be evoked with the filters UG3 and UG4T and not with the filters WG1 and GG3A. This was shown on several occasions in 2 subjects.

From these results it can be concluded that the delayed erythema and pigmentation in the methoxsalen-treated areas are probably elicited by wavelengths near 334 mμ and below 366 mμ.

No delayed erythema occurred in areas not treated with methoxsalen and exposed to sunlight through the filters UG3, UG4T, UG5, UG11, WG1, GG3A, GG14A and RG1A. As could be expected primary pigmentation was only seen through the filters UG3, UG4T, UG5, UG11, WG1, GG3A but not with GG14A and RG1A.

DISCUSSION

None of the substances applied *in the concentration used* altered the primary pigmentation. It appeared at the same time and in the same degree as in the controls. In comparing degrees of primary pigmentation one must realize that slight alterations of the angle of incidence of the sun's rays may produce great changes in pigmentation. Tronnier *et al.*[6] showed that some of these substances (15% hydrogen peroxide and 10% ascorbic acid in eucerin anhydricum) activated the phenomenon of secondary erythema and pigmentation. It should be noted that the so-called 'sunburn-protecting' ointments, such as 5% tannic acid and 5% sulfanilamide ointment, with absorption spectra between 290 and 320 mμ, did not prevent primary pigmentation.

It is remarkable that chlorpromazine, haematoporphyrin, and methoxsalen with absorption spectra in long-wave ultraviolet light also did not alter the primary pigmentation. According to the Grotthus-Draper Law, radiation must be absorbed in order to bring about a reaction.

An interesting feature is the finding of a secondary or delayed erythema with pigmentation the day following exposure to sunlight in the areas treated with methoxsalen — this was not noticed when the other substances were used. It also occurred after the sunlight had passed through a glass slide which cuts out the ordinary sunburn-producing rays of between 290 and 320 mμ. From the experiments with these filters it is concluded that this 'sunburn reaction' is probably caused by ultraviolet wavelengths near 334 mμ and below 366 mμ. It is generally accepted that sunburn-reaction is evoked by wavelengths between 290 and 320 mμ, although long-wave ultraviolet light can also produce it; then, however, much more radiant energy is necessary.

Several observations in the literature suggest that the psoralens are able to potentiate ultraviolet radiation in long-wave ultraviolet light. Sidi and Bourgeois-Gavardin[7] and also Fitzpatrick[8] reported activity through window-glass, Pathak and Fitzpatrick[9] reported it with Wood's light, while Fowlks *et al.*[10] recently showed a photosensitizing action of furocoumarins, e.g. methoxsalen, in bacteria in the presence of long-wave ultraviolet light. The exact wavelengths causing the reaction are not mentioned in any of these publications and it is possible that a few wavelengths below 320 mμ still reached the skin. It is conceivable that the psoralens strongly increase sensitivity to the wavelengths of the sunburn spectrum and that even these few wavelengths below 320 mμ could evoke a sunburn reaction.

Our findings show that this is not the case, but that wavelengths near 334 mμ produce the effect. A few wavelengths below 320 mμ are however transmitted through our filters UG3 and UG4T. A monochromator or an interference filter is needed to exclude these minimal transmissions below 320 mμ with certainty.

Assuming that this 'methoxsalen sunburn reaction' is caused by long ultraviolet wavelengths near 334 mμ, then we are probably dealing with a shift of the wavelengths producing the sunburn reaction.

Not much is known about the nature of the shift by psoralens but the study of this phenomenon might explain light sensitivity reactions. It is possible that in light-sensitivity conditions substances are formed in the skin which cause a shift in the sunburn reaction similar to that evoked by the psoralens. It is also possible that these substances make the skin more sensitive to long-wave ultraviolet light so that a 'sunburn reaction' could be evoked by long-wave ultraviolet rays.

In agreement with this view are the following findings. Wiskemann and Wulf[11] found, in cases of polymorphic light eruption, a pathological reaction due to long-wave ultraviolet light (320 - 400 mμ). In cases of porphyria[12] they found an erythematous reaction to wavelengths above 320 mμ. Readings were made 7 and 24 hours after exposure. Magnus, Porter and Rimington[13] produced abnormal reactions in a patient with porphyria cutanea tarda with a monochromator, e.g. swelling, ecchymoses and even blistering, sometimes leading to secondary pigmentation, telangiectasia, and atrophy at wavelengths of 400 mμ. This point is at the region of maximal absorption, the Soret band, of most porphyrins. This strongly suggests that in this patient photosensitivity was initiated by porphyrins in the skin. The skin reactions of this patient to ultraviolet light in the normal sunburn region of the wavelengths (around 300 mμ) were normal. However, Magnus *et al.*[13], in the case they report, deal with an early or immediate erythema evoked by wavelengths of 400 mμ in contrast to secondary erythema, observed with methoxsalen and caused by long-wave ultraviolet light.

Magnus and Porter[14] observed this early erythema immediately after exposure to irradiation while the secondary or delayed erythemata develop within about 6 hours. With the aid of a monochromator they determined the wavelengths to be between 360 and 390 mμ, but not at 400 mμ. In pathological conditions other wavelengths evoked an early erythema.

Scott and Molhuysen van der Walle[15] reported increased

sensitivity to the long-wave direct erythema-producing rays (heat rays) in patients with lupus erythematosus; in patients with dermatitis solaris more sunburn reactions as well as direct erythema reactions to long rays (transmitted through a glass slide) were observed than were seen in normal persons.

Riemerschmid and Quin[16] have demonstrated a correspondence between the visible absorption spectra of eosin and the chlorophyll porphyrin, phyllo-erythrin, and the regions of the spectrum which are most effective in producing lesions of photosensitization in the skin of merino sheep in South Africa. The above observations are consistent with the conception that photosensitivity reactions can be produced by wavelengths above 320 mμ due to certain substances laid down in the skin. The practical conclusion from this is that in most of the above cases the common sun-protecting agents are not effective, because they usually prevent only the wavelengths between 290 - 320 mμ from reaching the skin.

Finally it should be mentioned that Wiskemann[17] and Wiskemann and Wulf[11] found that most patients with polymorphic light eruptions, in contrast to healthy people, did not show direct pigmentation at wavelengths of 300 - 350 mμ. In another article Wiskemann and Wisser[18] described the examination of 192 healthy individuals in Hamburg for the occurrence of direct pigmentation with the aid of a xenon arc lamp and filters. They reported that of 110 people between 7 and 45 years of age 98% showed primary pigmentation and of 82 people between 45 and 79 years of age 72% showed this phenomenon. It should be noted that the difference in incidence of primary pigmentation found by Wiskemann and Wisser[18] and by us can be explained chiefly by the fact that they used a xenon arc lamp and we used sunlight. Nevertheless the findings suggest a connection between absence of primary pigmentation and photosensitivity.

SUMMARY

1. Local application of various substances, e.g. tannic acid, sulfanilamide, hydrogen peroxide, ascorbic acid, benoquin, haematoporphyrin, hormones, and methoxsalen, did not influence the primary pigmentation which can generally be produced by sunlight in all non-White people.

2. Protective agents for sunburn could not prevent primary pigmentation.

3. Although methoxsalen had no effect on primary pigmentation, the methoxsalen-treated area showed a sunburn reaction by the following day. With the aid of filters it was shown that this reaction was caused by long ultraviolet rays near 334 mμ and below 366 mμ. This 'shift' of wavelengths producing a sunburn reaction by methoxsalen is discussed with regard to photosensitivity reactions.

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We are indebted to the Upjohn Company, Kalamazoo, Michigan, USA, for supplying, free of charge, the crystalline methoxsalen used in these experiments.

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A CASE OF ACQUIRED HAEMOLYTIC ANAEMIA ASSOCIATED WITH PREGNANCY

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Acquired haemolytic anaemia associated with pregnancy is an uncommon occurrence, although there have been isolated reports of this condition. In the case presented here, an acute haemolytic crisis of a presumably acquired type made its first appearance in a pregnant woman and subsided shortly after delivery.

CASE REPORT

History

A patient, aged 28 years, was admitted to the Beilinson Hospital on 12 October 1958 in the fifth month of her first pregnancy. She complained of extreme weakness, palpitations, and respiratory distress. These symptoms had started a month earlier. A blood examination performed then had shown a haemoglobin of 7 g. per 100 ml. and a red cell count of 2,000,000 per c.mm. Intensive treatment with iron, vitamin B_{12}, and folic acid failed to improve her blood picture.

The patient was already known from 2 previous hospitalizations (1954, 1958) to be suffering from mitral stenosis and aortic insufficiency, proved by X-rays and cardiac catheterization. Blood examinations on both occasions were found to be within normal limits. At that time, conservative treatment produced a fairly good compensation.

On admission to the medical department, the patient displayed a marked pallor with no detectable icteric tinge. She had a severe orthopnoeic dyspnoea. Her blood pressure was 130/70 mm.Hg and her pulse-rate was 130 per minute. Her temperature was normal. There was no glossitis or brittle nails. The liver, spleen and lymph nodes were not palpable. The size of the uterus corresponded to the duration of the amenorrhoea. Auscultation of the heart confirmed the diagnosis of mitral stenosis and mild aortic insufficiency. Delicate râles were heard over the bases of both lungs.

Laboratory Investigations

The blood examination gave the following information: haemoglobin 4·6 g. per 100 ml.; red cell count 1,600,000 per c.mm.; haematocrit 18%; leucocytes 5,000 per c.mm.; platelet count 184,000 per c.mm.; reticulocytes 8·4%. The blood film showed marked anisocytosis with macro- and poikilocytosis,

moderate polychromasia and occasional nucleated red cells. Megalocytes and spherocytes were not found. The differential count was normal.

Bone marrow smears showed a striking proliferation of normoblasts without maturation abnormalities. Numerous haemosiderin-loaded macrophages were seen. The serum iron was 156 μg. per 100 ml. and the serum bilirubin 2·2 mg. per 100 ml. The 24-hour faecal urobilinogen excretion was raised on several occasions (350 - 500 mg.). Repeated examination for occult bleeding was negative. There was 1·2% alkali-resistant haemoglobin, but no electrophoretically abnormal components; osmotic and mechanical fragility of the erythrocytes was normal. The antiglobulin test (with the aid of papin-, fycin- and trypsin-treated cells), the Ham test and the Crosby test, cold agglutinins, and haemolysins, were all found to be negative. The urine and blood biochemistry was normal. Blood cultures remained persistently sterile. Haematological investigations of family members did not disclose any hereditary blood disorder.

Treatment and Follow-up

Conventional anti-anaemic treatment failed to prevent a rapid deterioration in the patient's condition. She was therefore given 3 transfusions of packed red blood cells (350 c.c. each) at short intervals. The haemoglobin went up to 8 g. for 2 days, then dropped rapidly and remained low despite 2 additional blood transfusions. It was then decided to attempt steroid therapy. The patient received 30 mg. meticorten daily for 3 weeks. At the end of that period, her haemoglobin rose to 8 g. per 100 ml. An increase of the daily dose of meticorten to 40 mg. resulted in an even more rapid and convincing response as the haemoglobin reached 11 g. per 100 ml. and the erythrocyte count 4,000,000 per c.mm. The faecal urobilinogen output and the reticulocyte count returned to normal. The general condition of the patient improved markedly. However, any attempt to maintain these therapeutic results with a smaller dose of meticorten caused a recurrence of the anaemia and an immediate increase in the faecal urobilinogen.

In the eighth month of her pregnancy, the patient gave birth by spontaneous delivery to a healthy and haematologically normal baby. Her haemoglobin dropped once more to 9 g. per 100 ml., due to loss of blood during the third stage of labour. She was given a transfusion of packed red cells and continued to receive meticorten in decreasing amounts. Two-and-a-half months after delivery her haemoglobin was again 12 g. per 100 ml. and the red cell count 4,000,000 per c.mm., allowing the ultimate withdrawal of the steroids with no further deterioration.

For a period of 9 months since this time the patient was under constant haematological supervision. Her blood picture remained normal, and she underwent a successful cardiac operation with no adverse effect on her blood condition.

DISCUSSION

The name 'haemolytic anaemia of pregnancy'[1] has not gained universal acceptance as describing a separate clinical and aetiological entity. This is probably due to the fact that various factors might be held responsible for an increased red-cell destruction in pregnant women. It is known that megaloblastic anaemia is associated with diminished viability of erythrocytes[2] which, in isolated cases, might be so pronounced as to simulate a primary haemolytic syndrome. Acute haemolytic episodes have been described in late pregnancy or early in the puerperium.[3] A sudden onset with fever, leucocytosis, reticulocytosis and a megaloblastic type of anaemia are their main features. These cases have been identified as haemolytic anaemia of the Lederer type, a controversial entity, frequently mentioned in the literature. They respond promptly to single blood transfusions and liver therapy. These cases might very well be megaloblastic anaemias with exaggerated haemolysis resulting from some intercurrent infection.

Still more confusing are cases of primary haemolytic anaemia in pregnant women with megaloblastic transformation due to secondary vitamin B_{12} or folic acid depletion.[5] This variety of anaemia is not improved by the administration of the deficient haemopoietic factors, although a return to normocytosis has been observed.[6]

Difficulties may also arise when trying to distinguish between congenital and acquired types of haemolytic anaemia in pregnancy. It is usually accepted that the diagnosis of the congenital type requires the presence of familial features and signs of corpuscular defect, such as spherocytosis, sickling, increased fragility of the erythrocytes, abnormal haemoglobins, etc. Yet, this is not always the rule. Cases of latent congenital haemolytic anaemia are known to appear for the first time in adult life, and in the absence of a positive family history.[7] Association of such instances with pregnancy have also been described.[8] Isolated spherocytes or a mildly-increased osmotic fragility may be encountered in acquired haemolytic anaemia, whereas the antiglobulin test has been reported positive in definite cases of the congenital type.[9]

The recognition of the acquired mechanism of a haemolytic anaemia usually rests on positive serological evidence. In cases in which no immune factors are detected, survival studies of transfused normal red cells have been used, and decreased viability, whenever found, has been taken as proof of an extracorpuscular mechanism of the haemolysis.[6]

The clinical approach might be useful in discerning haemolytic anaemia secondary to some underlying disease such as leukaemia, malignant lymphoma, systemic or infectious disease or exposure to injurious agents, whose association with pregnancy is a potential possibility. Haemolytic anaemia may accompany complications due to the pregnancy proper, such as eclampsia or toxaemia of pregnancy.[10-13] A case of haemolysis associated with toxaemia in 2 consecutive pregnancies has been reported.[14]

When all these considerations are taken into account, only a few cases of an acquired anaemia appearing in a normally proceeding gestation with no detectable signs of some concomitant disease may be found in the literature. In these rare cases, the anaemia completely subsided[15] or was greatly improved after delivery.[6,16]

In the case presented here, we find all the necessary criteria of an acute haemolytic syndrome of the acquired type. There was a sudden and rapidly-progressing anaemia in a woman whose personal and familial haematological background, confirmed by the appropriate investigations, could not disclose any hereditary disorder. Neither could evidence of toxaemia or exposure to injurious agents be detected. No underlying disease liable to promote haemolysis could be found. The anaemia was associated with definite signs of increased red-cell destruction, such as elevated serum-iron and bilirubin levels, increased excretion of faecal urobilinogen, peripheral reticulocytosis, and normoblastic proliferation in the bone marrow. Furthermore, repeated apparently compatible transfusions failed to achieve any lasting improvement of the anaemic condition, and an immediate increase of the faecal urobilinogen output resulted. This pointed to an indiscriminate destruction of the donor's as well as the patient's red cells.

hence to an extracorpuscular reason for the haemolysis. The anaemia responded favourably to steroid therapy, as anaemias of auto-immune origin usually do, although repeated serological investigations failed to disclose the presence of immune haemolytic factors of any type. The negative serological results in this particular type of acquired haemolytic anaemia in pregnant women have been recognized in all the abovementioned reports. The observation of the favourable effect of steroid therapy has been confirmed.[6]

An interesting feature of this anaemia was its association with gestation and its rapid subsidence after delivery. The relationship between haemolysis and pregnancy, whether incidental or precipitating, has been frequently debated.[17,18] However, no one has been able to advance any definite proof of a possible immunological or hormonal mechanism,[19] and the problem still awaits further elucidation.

SUMMARY

A case of acquired haemolytic anaemia associated with pregnancy is described. The use of meticorten brought the haemolysis under control. Only after delivery did the blood picture return to normal without steroid treatment. Aspects of the diagnostic problem of acquired haemolytic anaemia during pregnancy are discussed.

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CRYPTOGENIC HEART DISEASE IN THE BANTU OF SOUTH AFRICA*

A meeting of the Association of Physicians of South Africa (M.A.S.A.) was held at Baragwanath Hospital, Johannesburg, on 9 July 1960.

Dr. V. H. Wilson, senior physician at Baragwanath Hospital, as Chairman of the meeting, welcomed Dr. M. M. Suzman, Dr. S. J. Fleishman, and 30 members of the Association of Physicians. He stated that up to 400 cases of cryptogenic heart disease (CHD) per year were admitted to Baragwanath Hospital, and added that this disease still defied explanation.

The Problem

Dr. K. J. Keeley (Baragwanath) presented the aetiological spectrum of heart disease as seen in the wards. Cryptogenic heart disease forms 37·5% of the total, outstripping chronic rheumatic heart disease (20·3%) and hypertensive heart disease (19·6%). Males constitute 64·0% of the cryptogenic cases.

Clinical Presentation

Dr. H. C. Seftel, of Baragwanath Hospital, outlined the clinical presentation of cryptogenic heart disease: The condition affects both sexes with a male predominance of 1·6 : 1. It occurs in all age groups, including children under 10 years, but the maximum age incidence for both sexes is between 30 and 50.

In respect of tribal origin, occupation, educational status, diet, alcohol and tobacco consumption, and the use of witch-doctor preparations, the patients with CHD appear to be essentially similar to the African population at large.

Clinically, the condition presents with heart failure, both left-sided and right-sided. The circulation is hypokinetic or normokinetic and sinus rhythm is usual. The blood pressure is often elevated on admission, but this is transient, returning to normal within a week or two. The heart is always enlarged, often markedly so. A heaving impulse is frequently felt over the left or right ventricle. Gallop rhythm is almost invariable. In many cases functional mitral or tricuspid systolic murmurs are heard. The great majority of cases show no clinical stigmata of malnutrition.

Electrocardiographically the majority of cases show increased voltage of the QRS complex and inverted T waves over the left ventricle.

With bed rest, digitalis, and diuretic therapy the signs of heart failure and gallop rhythm disappear and the systolic murmurs diminish in intensity. Cardiomegaly, however, usually persists and at this stage (about 1 - 2 weeks after admission) the patients are discharged from hospital. Our knowledge of the subsequent course is incomplete, no systematic follow-up study having yet been undertaken. The general view, based entirely on hospital experience, is that many, if not most, cases relapse and that with each relapse the condition becomes increasingly refractory to therapy.

There is, however, a group of patients with CHD whose course has been studied in some detail, and has been shown to be quite different from that described above. This group consists of patients in whom the onset of heart failure occurs in the postpartum period, or less often, in the last trimester of pregnancy. These peripartum cases are not uncommon, constituting about one third of all cases of CHD in females. They tend to be women of high parity and high child-bearing age, who have a higher-than-normal incidence of twin pregnancies.

These cases are clinically similar to the general run of cases with CHD except that in half the cases recovery ensues. This may occur within a few weeks, but usually requires between 3 and 12 months. Recovery may occur despite the cessation of digitalis and diuretic therapy and the resumption of normal physical activity while the heart is still enlarged. The other half pursue a chronic or relapsing course. The total mortality rate appears to be about 25%. Prognosis is significantly related to high age, high parity, and persistent lactation after the onset of heart failure. The condition may recover completely only to recur after a subsequent pregnancy.

Course of Illness

Dr. Keeley illustrated the possible course of the illness with selected cases. Death rarely results from the first attack of congestive failure; the number of patients who recover completely from the first attack is not known, because many subjects do not return after recovery from the congestive state. Usually there is a steady progression, punctuated by repeated admissions in congestive failure, until death ensues. By contrast, one patient was known to have had a complete clinical and radiological remission for at least 19 months after having been in congestive failure intermittently for 4½ years.

Radiology

Dr. N. Joffe, of Baragwanath Hospital, said that in patients who are X-rayed during an acute phase of the disease, be this the first episode of cardiac failure or a recurrence, the most constant radiological finding is the presence of cardiomegaly

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which varies from a slight increase in the transverse diameter of the heart to a gross degree of enlargement. Both the left and right sides of the heart are involved and, when the cardiomegaly is of a marked degree, the radiological appearances are not unlike those of a pericardial effusion. Although biventricular enlargement is the rule, predominance of the left ventricle is not uncommon. The aortic shadow shows no abnormality. Slight enlargement of the main pulmonary artery segment and pulmonary vascular engorgement are common findings while the patient is in cardiac failure; pulmonary oedema is rarely observed. Small unilateral or bilateral pleural effusions are frequently demonstrated, but large effusions are uncommon.

In the majority of cases fluoroscopic examination of the chest discloses a diminution in the amplitude of cardiac pulsations and, rarely, an almost complete absence of pulsations may be observed. In a fair proportion of cases, however, cardiac pulsations appear to be of normal amplitude. Pulsation of the aorta is commonly of poor amplitude.

The presence of associated primary lung disease is not common. Acute pulmonary infections and tuberculosis do not appear to be more frequent than in the average Bantu population; pulmonary infarcts are not infrequent in this disease and may be demonstrated radiologically by the presence of one or more pulmonary opacities which may be found in any situation in the lung fields. These opacities may be unilateral or bilateral in distribution and are occasionally accompanied by small pleural effusions or elevation of the corresponding dome of the diaphragm.

Recovery from the acute episode of cardiac failure is usually accompanied by radiological evidence of some reduction in heart size and an improvement in the amplitude of cardiac and aortic pulsations.

Pathology

Dr. C. Isaacson, of the South African Institute for Medical Research and Baragwanath Hospital, stated that this information had been obtained from 537 consecutive cases of heart failure in a total of 2,367 necropsies; the cases of heart failure thus formed 22·7% of all causes of death. Cryptogenic heart failure is the fourth commonest cause of cardiac failure in the Bantu in Johannesburg as seen at necropsy. It is probable that the incidence is even higher, since other cases of heart failure were observed, such as hypertensive heart failure in which the lesions present seemed inadequate to account for failure. The corrected sex ratio showed a male preponderence of 1·6:1.

Macroscopic examination showed that the heart was hypertrophied and dilated in all cases. The hypertrophy was usually generalized and involved all chambers equally, but in 3 cases the enlargement was more marked on the right side. In 30 hearts no other significant lesions were observed. In 48 hearts intraluminal thrombi were present. These varied from recent mural thrombi superimposed on an apparently intact endocardium and myocardium to dense organized thrombi forming fibrous plaques obliterating the spaces between the trabeculae carneae. In 2 hearts thick, fibrous plaques were present overlying the endocardium. From the microscopic examination it is believed that these represent old organized thrombi. In only 1 heart was the extent of the endocardial fibrous plaques comparable to that described in Uganda. No significant valvular lesions were seen in any case. In some hearts the coronary arteries showed mild atherosclerosis. In no case was significant diminution of the lumen present and the degree of atherosclerosis was no greater than that seen in hearts of patients dying from non-cardiac disease. Microscopic examination of the endocardium showed it to be normal in the majority of hearts with only occasional areas of non-specific diffuse and focal thickening. In some cases small foci of infiltration by lymphocytes, macrophages, and plasma cells were noted. In some hearts large bulky recent thrombi could be seen deposited on an apparently normal endocardium. In other areas the thrombi were composed of very small fibrin clots. The thrombi were most frequent between the interstices of the trabeculae carneae. The myocardium consistently showed hypertrophy of the muscle fibres which contained large 'stag-horn' nuclei. Sometimes the fibres showed a mild non-specific intracellular oedema with separation of myofibrils. No parasites or significant haemosiderin deposits could be demonstrated. In many hearts there was some degree of interstitial oedema between the muscle fibres associated sometimes with a deposition of a poorly-staining collagenous tissue. Small foci of fibrous scarring were sometimes observed with degeneration of muscle fibres. Such scarring was usually mild but was more prominent in 17 cases. Such areas of scarring did not imply coronary ischaemia, since the coronary vessels were normal. In no case did the small myocardial arteries and arterioles show evidence of fibrinoid necrosis or an arteritis. Examination of the other organs showed generalized venous congestion and in addition in 28 cases there was evidence of visceral infarction. The kidneys showed only the normal changes associated with increasing age.

The findings in the hearts of children dying of cryptogenic heart failure were essentially similar to those observed in adults. The endocardium of the left ventricle in these cases showed perhaps more fibrous thickening than in adults.

Points in Discussion

While the cases of CHD seen at the Baragwanath Hospital were mostly in urbanized Africans, Dr. J. V. O. Reid (Durban) and Dr. M. Nellen (Cape Town) stated that they had observed the condition in rural Africans as well. Dr. Reid had also encounted peripartum cases of CHD. His findings in these and in patients with CHD in general were similar to those of the Baragwanath Hospital workers.

Several speakers raised the problem of differentiating CHD from pericarditis. Dr. Wilson thought that if a heaving or thrusting impulse was felt over the praecordium, pericarditis was most unlikely. He stressed the danger of attempting to distinguish the two conditions by pericardial paracentesis. Dr. Reid agreed and advocated right atrial catheterization to assess the thickness of the atrial wall. He also stated that while pulsus paradoxus might occur in both conditions, a marked degree of paradoxus with disappearance of the pulse on inspiration favoured the diagnosis of pericarditis. Dr. Nellen stated that the finding of quadruple rhythm strongly suggested CHD. He had never found this rhythm in pericarditis.

Dr. M. M. Suzman (Johannesburg) raised the question of the relationship of CHD to beri-beri. Dr. Wilson reported that beri-beri was seen at the Baragwanath Hospital but, by contrast with CHD, was a rare disease. Patients with CHD did not have a hyperkinetic circulation, they were not influenced by thiamine and they were not usually rapidly reversible.

Dr. S. Fleishman (Johannesburg) asked whether cortisone had been used in CHD. Drs. Reid, Keeley, and Seftel had tried cortisone or its analogues but without success. Dr. Seftel mentioned that an attempt had been made to detect cardiac antibodies in the serum of cases of CHD by the agar-gel diffusion and tanned, red-cell techniques. The results were negative.

Dr. Suzman thought that CHD might have been diagnosed as hypertensive heart disease in the past.

Dr. L. Klugman (Johannesburg) suggested that the hypertrophy of the heart in CHD was due to the diastolic overload in a dilated heart. Dr. Isaacson considered that it had been satisfactorily demonstrated that the fibrous endocardial thickening seen in any heart failure was a mechanism to compensate for the increased tensile load placed on the myocardium of a dilated heart.

Dr. C. F. van der Merwe (Pretoria) asked what the autopsy findings were in cases of CHD in which recovery had ocurred and death was subsequently due to some unrelated cause. Dr. Seftel quoted a single case in which clinical, radiological and electrocardiographic recovery had occurred, the patient dying 2 months later from infective hepatitis. The heart still showed hypertrophy on postmortem examination.

Dr. Wilson, as Chairman, urged the meeting to accept that the cause of CHD was still unknown and warned that in attempting to find a stereotyped pathological explanation an important factor in myocardial dysfunction might be overlooked.

ROYAL NAVAL HOSPITAL, CAPE OF GOOD HOPE

REPORT OF TWO CASES OF CAPITAL OPERATIONS BY STAFF SURGEON-IN-CHARGE, JOHN BERNARD, R.N., 24 MAY 1873

CASE I

Necrosis of the lower third of the right femur, with destruction of the knee joint. Amputation of the thigh. Recovery.

History

William Nash, about 20 years of age, Ordinary Seaman, H.M.S. 'Narcissus', and of a scrofulous constitution, was sent from his ship to the military hospital, island of Mauritius, on 5 June 1872 for disease of the right femur and knee joint. On 10 September he was discharged from the military hospital, island of Mauritius, to H.M.S. 'Himalaya', and by her conveyed to the Royal Naval Hospital, Cape of Good Hope, where he was admitted on 25 September 1872.

Condition on Admission

25 September. On admission the right knee joint was greatly enlarged and fluctuation could be distinctly felt in it. About 4 inches above the joint on the inner side of the thigh, there was a sinus which discharged thin pus freely and which led down through a large cavity to bare bone, at the lower part, and to roughened bone at the upper part. The patient was greatly emaciated and there was hectic fever.

A few days after admission an incision was made into the knee joint which gave exit to a considerable quantity of pus mixed with synovial fluid. The joint and the sinus which led down to the diseased bone were well syringed with solution of carbolic acid (1 in 60). The patient was placed on as good a diet as his stomach was capable of retaining.

2 October. In consultation today it was decided to amputate the limb as the only means of saving the patient's life, it having become daily more evident that, owing to the increased discharge of pus, and hectic fever, death must ensue if any further delay occurred in trying to save the limb.

The operation by means of an anterior and posterior flap at the junction of the upper and middle third was accordingly performed this day under chloroform. Solution of carbolic acid in water was applied to the flaps by syringing during the operation. The flaps were brought into apposition and retained *in situ* by silver wire sutures, scraped lint steeped in solution of carbolic acid being applied around the edges of the flaps.

18 October. The patient has progressed in a most satisfactory manner since the operation, no unfavourable symptom having occurred — all the ligatures came away this day. The flaps have united by 'the first intention' in all their extent except at the inner and upper angle through which the ligatures protruded. His general health is greatly improved, and he is allowed to get up.

15 November. The stump is quite healed. The patient's general health is re-established and he has been going about on crutches for the last fortnight. He was invalided this day.

24 April 1873. Discharged to H.M.S. 'Barrosa' for a passage to England.

Examination of the Diseased Limb

On examination a large cavity containing pus was found in the lower and back part of the thigh and popliteal space. This communicated with the knee joint behind the posterior ligament. The lower extremity of the femur was denuded of periosteum for about 4 inches and was of a dirty green colour above the inner condyle. Above this the bone was roughened and in a carious state. The tissues (*sic*) around the knee joint were thickened, the cartilages ulcerated, and the inner condyle of the femur in a carious state. The inner side of the head of the tibia was bare exposing the cancellous (*sic*) tissues. The leg was oedematous.

CASE II

Necrosis of the left humerus with disease of both shoulder and elbow joints. Amputation at the shoulder joint. Recovery.

History

George Davis, about 21 years of age, Ordinary Seaman, H.M.S. 'Glasgow', was sent from his ship to the military hospital, island of Mauritius, on 4 July 1872 for acute inflammation of the left shoulder joint followed by suppuration and caries of the bone.

On 10 September he was discharged from the military hospital, island of Mauritius, to H.M.S. 'Himalaya' and by her conveyed to the Royal Naval Hospital, Cape of Good Hope, where he was admitted on the 25 September 1872.

Condition on Examination

25 September. On admission several sinuses copiously discharging pus were observed leading down to dead bone, both on the anterior and posterior aspects of the arm. Both shoulder and elbow joints were found to be extensively diseased, and the forearm and hand were oedematous and livid. There was much emaciation and hectic fever.

7 October. It was determined in consultation to amputate at the shoulder joint as the only means left to save the patient's life. This operation was accordingly performed this day under chloroform, an external flap being made by dissecting up the deltoid, then disarticulating and forming an internal flap. Owing to the head of the bone being found separated at the surgical neck from the shaft, it had to be carefully dissected out, without the advantage of the arm being used as a lever.

About 3 hours after the operation, while the patient was retching and vomiting, violent haemorrhage occurred from the stump. The subclavian artery was at once controlled by means of a rag (*sic*) and the stump opened and a ligature applied to the bleeding vessel, a branch of the circumflex, which had been twisted during the operation. The flaps were well syringed with a solution of carbolic acid in water and brought into apposition and retained *in situ* by silver wire sutures; lint steeped in the same solution being applied around the edges.

25 October. The patient has progressed favourably since the operation, the flaps are almost entirely united by 'the first intention', but there is a glazed and inflamed appearance at the end of the internal flap where fluctuation is distinct. His strength and general health are much improved. An incision was made into the abscess and exit given to a quantity of pus. The opening was freely syringed with carbolic acid in water and dressed with lint steeped in the same solution.

31 October. The patient has progressed most favourably since the last report. The abscess is quite healed. The flaps, excepting a small portion on the anterior part where the ligatures protruded, are united by 'the first intention'. Some of the ligatures came away yesterday, and the remainder have come away today. The patient's general health is re-established and he is able to get up and walk about.

15 November. The stump is quite healed. The patient was brought forward for survey this day and invalided.

24 April 1873. Discharged to H.M.S. 'Barrosa' for passage to England.

Examination of the Diseased Limb

The head of the humerus was found separated from the shaft at the surgical neck. There was a cavity about the size of a filbert in the head, which was carious externally.

On being sawn perpendicularly, the shaft was found to be dead and partly detached from the new bone which had been thrown out around and enclosed it. The elbow joint was found extensively diseased, and the heads of the radius and ulna were affected.

POSTSCRIPT

The reports of the two operations performed at the Royal Naval Hospital, Simonstown, in 1872, are of great interest as far as the history of medicine in this country is concerned. The owner of the manuscripts is Mrs. R. E. H. Amman, (neè Bernard) of St. James, Cape, who is a granddaughter of Dr. John Bernard. The manuscripts were brought to the attention of Prof. J. H. Louw, Professor of Surgery, University of Cape Town, (who submitted them for publication in the *Journal*) by Mr. H. C. Willis of Simonstown. Mr. Willis received them on loan from the Simonstown Historical Society. The information about the surgeon, John Bernard, was obtained from

his various medical certificates and extracts from a publication *The Bernards of Kerry* by G. H. Bernard (University Press, Dublin, 1932).

John Bernard belonged to a family which produced several naval surgeons, some of whom achieved a very high rank. His father was a naval surgeon and so were two of his older brothers, Robert and Sidney. His father, also John Bernard, received the M.D. degree at the University of Edinburgh in May 1817. He died in 1851.

His brother, Robert, was greatly impressed by Lister's antiseptic treatment of wounds and made a special study of the technique in London hospitals. It is believed that he was the first to introduce Lister's method into naval hospitals, and his work in this connection attracted much attention when he was appointed head of the surgical side of the Royal Naval Hospital at Stonehouse in 1868. In view of the fact that he was responsible for spreading Lister's doctrines in Royal Naval hospitals throughout the world, it is of interest that he died of septicaemia following an infection of his finger in 1872.

A cousin, Sidney Bernard, was also a naval surgeon and was actually assistant surgeon at the Cape in 1844, when he served in H.M.S. 'Melville Castle'.

John Bernard was born in 1823. He received his medical education in Dublin, obtaining his various certificates from the Royal College of Surgeons of Ireland between 1837 and 1841. At the age of 18 years he became assistant surgeon in the Royal Navy in October 1841. He seems to have been at the Cape *circa* 1865 - 1873 or 1870 - 1875, when he was appointed Deputy Inspector General and later Inspector General. He was naturally greatly influenced by his older brother's teachings on antiseptic surgery and it would appear that the two successful operations, which have been so carefully documented, were the first to be performed in South Africa according to Lister's methods. In this connection it is interesting to note that Lister's paper on antiseptics was published in 1867, i.e. 5 years before these operations were performed.

WORLD HEALTH ORGANIZATION

EMERGENCY HEALTH ACTION IN CONGO

Countering Serious Epidemiological Threat

Emergency action is being taken on the health side of the United Nations operation in the Congo, in order to avert the possible consequences of the breakdown of preventive health services in many parts of the country. The immediate target is to recruit 100 medical and highly skilled technical staff, principally to reactivate the preventive health services designed to stop the spread of communicable diseases.

In reporting on the deteriorating situation to the United Nations and to WHO headquarters in Geneva, Dr. J. S. McKenzie Pollock, Senior WHO Representative in the Congo, warned that the momentum of the past is now slowing down, and that the effects of the discontinuation of preventive measures are now being seen. In the course of one recent week, 4 separate outbreaks of smallpox were reported, and it is possible that other outbreaks are present but, because of inadequate communications, have not been notified. As they stand at present, the public-health services of the country are not in a position to combat this serious epidemiological threat. Stocks of vaccine are available, thanks to the generosity of the Nigerian Government, but the technical skill necessary to organize a large-scale vaccination programme is lacking, and such a programme can only be started when technical staff from outside can be made available to guide the Congolese public-health services and give leadership to the local field health teams.

Smallpox and Plague

The threat is not confined to smallpox. Dr. McKenzie Pollock stated that, while the smallpox situation gives dramatic indication of the breakdown in the health services, other disease patterns are also showing significant deterioration. In the Bunia area, for instance, an outbreak of bubonic plague is at present being investigated. Its extent is as yet unknown, but some deaths have occurred. In addition, disquieting reports are being received of an increased number of malaria and filariasis cases while, Dr. McKenzie Pollock warns, if preventive measures are not restarted, an increase in the incidence of sleeping sickness can also be expected.

The WHO advisory team to the Central Ministry of Health has drawn up a list of doctors and senior technicians needed to reactivate the public health services of the Congo. Under the emergency action sanctioned by the United Nations, which will involve an expenditure of several million dollars, the priority target is for recruitment immediately of about 100 qualified staff to deal with the epidemiological situation and, subsequently, to obtain the services of another 300 - 400 medical and technical personnel, which it is hoped to recruit before the end of the year.

More Red Cross Teams Needed

By the time this total is reached, not only the preventive health services, but also the medical care services, including hospital and rural health services, can become fully operative, even after the Red Cross and Red Crescent, as well as government medical teams, have been withdrawn. The services of most of these were made available for a period of 3 months only and, in many instances, half of this period has already expired. At the peak point, a total of nearly 160 qualified medical and technical personnel, divided into 33 teams and representing 22 countries, manned the hospital services of the Congo. However, the USSR team of 20 doctors and 4 interpreters, as well as the Israeli team of 8 doctors and 5 nurses and technicians, have now been withdrawn. As an interim measure, 5 further Red Cross teams are being called upon for service in the Congo by the League of Red Cross Societies, at the request of WHO.

The new recruitment drive for medical personnel is the first large scale one of its kind to be launched by the United Nations Operation in the Congo. The medical personnel will be under contract to the Congolese Government, but will enjoy the protection of the United Nations, as well as a salary guarantee until budgetary arrangements between the United Nations and the Congolese Government have been worked out. The recruitment services of the World Health Organization will be used for the drive, and will, *inter alia*, ensure that adequate standards of technical competence are maintained. It is hoped that, in the first instance, personnel can be recruited from neighbouring countries with disease patterns similar to those of the Congo.

OFFICIAL ANNOUNCEMENT : AMPTELIKE AANKONDIGING

MEDICAL AID SOCIETIES

Members of the Association are requested to note the change of name of certain medical aid societies as set out hereunder:

1. Corner House Insurance Fund to Corner House Medical Aid Fund.
2. New Consolidated Goldfields Employees' Medical Aid Fund to Goldfields Medical Aid Fund.

L. M. Marchand
Associate Secretary

28 - 31 Plaza Building
Pretoria
4 October 1960

MEDIESE HULPVERENIGINGS

Lede van die Vereniging word versoek om te let op die naamsveranderinge van sekere mediese hulpverenigings soos hieronder vermeld:

1. Corner House Insurance Fund na Corner House Medical Aid Fund.
2. New Consolidated Goldfields Employees' Medical Aid Fund na Goldfields Medical Aid Fund.

L. M. Marchand
Medesekretaris

Plaza-gebou 28
Pretoria
4 Oktober 1960

PHARMACEUTICAL NEWS : FARMASEUTIESE NUUS

NEW MEDICAL FILMS

SKF Laboratories have available on loan, without charge, a film entitled 'Recognition and management of respiratory acidosis', which is intended for showing to professional audiences only. It is a 16 mm. sound film in colour and the running time is 35 minutes.

This teaching film presents a clinic on a topic of increasing interest to the medical profession. The course of a fatal case of respiratory acidosis is illustrated and discussed by the panel. The aetiology and symptoms of this condition are examined in detail and the importance of early recognition emphasized. A suggested treatment programme, including emergency treatment of acute cases, is outlined. The use of various types of pressure-breathing apparatus is demonstrated with patients.

Bookings for the film may be arranged through local SKF representatives or by writing to SKF Laboratories, P.O. Box 38, Isando, Transvaal. If possible, 4 weeks' prior notice and an alternative showing date of at least 1 month after the preferred date should be given.

* * *

British Schering Ltd. have recently announced the release of a new film entitled 'Thyrotoxicosis and thyroidectomy', which is available on loan to the medical profession. It is a 16 mm. colour film with sound commentary and the running time is 21 minutes.

The film illustrates the modern techniques used in the diagnosis and treatment of thyrotoxicosis, and the progress of one thyrotoxic patient is followed from the initiation of the preliminary investigation until the final check-up after surgery. 'Thyrotoxicosis and thyroidectomy' was filmed at the New End Hospital, London, where there is a specialized thyroid unit fast becoming recognized as a centre for the study of techniques in the treatment of thyroid disease.

Westdene Products (Pty.) Ltd., who are the distributors for British Schering in South Africa, will supply this film on loan to interested persons.

Other films available on application to Westdene Products (Pty.) Ltd., P.O. Box 7710, Johannesburg, include: 'PAS', 'A simple method of cataract extraction', 'Adrenosem—osteopathy of the mandible for the correction of prognathism', 'A hundred million electron volts against cancer', and 'Medicine in the tropics'.

PASSING EVENTS : IN DIE VERBYGAAN

American College of Chest Physicians, 1961 Prize Essay Contest. The American College of Chest Physicians is offering three cash awards to winners of the 1961 Prize Essay Contest. First prize $500.00, second prize $300.00, third prize $200.00. Each winner will also receive a certificate. The contest is open to *undergraduate* medical students throughout the world. Essays may be written on any phase of the diagnosis and treatment of chest diseases (cardiovascular or pulmonary). The contest closes on 1 April 1961. For applications and further information please write to the American College of Chest Physicians, 112 East Chestnut Street, Chicago 11, Illinois, USA.

* * *

Nutrition Society. The British Nutrition Society will hold a meeting at the Middlesex Hospital Medical School, London, W.1, on Saturday 17 December 1960 on 'Calorie balance in Man', which may be of interest to practitioners proceeding overseas at that time. Sir Charles Dodds, F.R.S., will be the Chairman of the meeting and among the subjects to be discussed are 'Physiological principles', 'Environmental factors', 'Endocrine factors', 'Fat-mobility factor', 'Neurological and psychological factors', 'Calorie balance during pregnancy and lactation' and 'Energy balance in early life'. Further information may be obtained from the Hon. Programmes Secretary, D. F. Hollingsworth, c/o Ministry of Agriculture, Fisheries and Food, Great Westminister House, Horseferry Road, London, S.W.1.

* * *

University of Cape Town and Association of Surgeons of South Africa (M.A.S.A.), Joint Lectures. The next lecture in this series will be held on Wednesday 26 October at 5.30 p.m. in the E-floor Lecture Theatre, Groote Schuur Hospital, Observatory, Cape. Mr. B. Shandling will speak on 'Unusual aspects of Hirschsprung's disease'. All members of the Medical Association are welcome to attend this lecture.

* * *

South African Institute for Medical Research, Johannesburg, Staff Scientific Meeting. The next meeting will be held on Monday 31 October at 5.10 p.m. in the Institute Lecture Theatre. Dr. S. Klempman will speak on 'Sex chromosomes and intersexes'.

* * *

Nagraadse Skool Beplanningskomitee, Bloemfontein. Die naweekkursus van 23 - 25 September 1960 het besonder goed verloop. Dertig kollegas, meerendeels van Bloemfontein, maar 'n hele paar van die platteland, het vir die kursus ingeskryf. Een kollega van Noupoort het ook gekom.

Prof. A. J. Brink, van die Universiteit van Stellenbosch, en prof. F. G. Geldenhuys van die Universiteit van Pretoria, en saam met hom dr. E. W. Sonnendecker van die Departement van Ginekologie, het elkeen 'n bydrae gelewer. Hulle het 'n groot deel van die verrigtinge bygewoon terwyl plaaslike kollegas hulle voordragte gelewer het, en het ook vrylik aan die besprekings deelgeneem.

Die gehalte van die voordragte was deurgaans goed en al die kursusgangers het almal gunstige kommentaar daaroor gelewer.

Saterdagaand was daar 'n geslaagde skemerkelkparty by die Bloemfonteinklub en almal het die geleentheid gehad om saam met hulle eggenotes gesellig met mekaar te verkeer.

* * *

Mr. Norman Petersen, F.R.C.S. (Eng.), F.R.C.S. (Edin.), of Cape Town is retiring from private practice at the end of October, but will be continuing his connection with Groote Schuur Hospital and the Red Cross War Memorial Childrens' Hospital.

* * *

National Cancer Association of South Africa. It has come to the knowledge of the National Cancer Association of South Africa that certain unauthorized persons are collecting funds for cancer or cancer research, purporting to act on behalf of the Association. Would-be donors are enjoined to request canvassers seeking donations to exhibit their authority from the National Cancer Association of South Africa before making contributions. Collection lists issued by the Association are easily identifiable, since they are printed and numbered (and not typed or roneoed) and bear the Association's emblem and the name of the Association printed in both official languages. In addition each form contains the name of the person authorized to collect, together with the signature of the official empowered to authorize such collection. Similarly, receipts issued by the National Cancer Association of South Africa, in respect of donations received, bear the name of the Association in bold print.

Nasionale Kankervereniging van Suid-Afrika. Dit het die Nasionale Kankervereniging van Suid-Afrika ter ore gekom dat sekere ongemagtigde persone wat voorgee dat hulle namens die Vereniging optree, geld vir kanker of kankernavorsing insamel. Lede van die publiek wat van plan is om bydraes te doen, word dringend versoek om die persoon wat om 'n donasie by hulle kom aanklop, te vra om sy of haar magtiging van die Nasionale Kankervereniging te toon voordat hulle enige geld oorhandig. Kollektelyste uitgereik deur die Vereniging kan maklik herken word, want hulle is gedruk en genommer (nie getik of afgerol nie), en hulle dra die

wapen en die naam van die Vereniging, gedruk in albei landstale. Op iedere vorm verskyn ook die naam van die persoon wat gemagtig is om geld namens die Vereniging in te samel, sowel as die handtekening van die amptenaar wat sodanige kollekte gemagtig het. Op dieselfde wyse verskyn die naam van die Nasionale Kankervereniging van Suid-Afrika in groot druk op alle kwitansies wat deur die Vereniging ten opsigte van donasies uitgereik word.

* * *

Dr. J. N. de Klerk, uroloog, van Kaapstad, wat 'n paar maande lank nie met sy praktyk kon voortgaan nie weens ongesteldheid, het nou herstel. Hy sit sy praktyk weer soos gewoonlik voort te Mediese Sentrum, Heerengracht, Kaapstad.

NEW PREPARATIONS AND APPLIANCES : NUWE PREPARATE EN TOESTELLE

KENACOMB OINTMENT

Squibb Laboratories (Pty.) Ltd., announce the introduction of Kenacomb Ointment, and supply the following information:

Since triamcinolone acetonide, a potent new corticosteroid for topical dermatological use was developed by the Squibb Institute for Medical Research, Squibb Laboratories have made available to South Africa a number of useful dermatologic preparations.

The first presentation to arrive in South Africa in April 1959 was Kenacort-A Ointment (Squibb triamcinolone acetonide) especially intended for inflammatory, pruritic and allergic skin conditions when bacterial superinfection is not a problem.

In the same month a Kenacort-A preparation with protection against bacterial superinfection became available. This is Kenacort-A with Graneodin Lotion. Graneodin (Squibb neomycin-gramicidin) is effective for the treatment and prevention of many superficial bacterial infections of the skin. Kenacort-A with Graneodin Lotion is especially suited to wet, weeping lesions. For dry, scaly skin conditions, Kenacort-A with Graneodin Ointment was introduced in December 1959.

In April 1960 Kenacort-A with Graneodin Ophthalmic Ointment was made available offering prompt, gratifying, and usually prolonged relief of inflamed, itching and burning eye conditions.

Squibb Laboratories are now adding a new, even more useful preparation to the already established Kenacort-A family of products. This preparation is marketed under the trade name Kenacomb Ointment.

Each gramme of Kenacomb Ointment supplies: triamcinolone acetonide 1·0 mg., neomycin base (as sulphate) 2·5 mg., gramicidin 0·25 mg., and nystatin 100,000 units.

Kenacomb Ointment exerts 5 distinct therapeutic actions in one preparation: anti-inflammatory, antipruritic, anti-allergic, antibacterial and antifungal. It is especially formulated to prevent or combat bacterial or monilial infection in cutaneous disorders. Topically applied, Kenacomb controls and induces a rapid clearance of burning and itching inflamed skin lesions — and provides safe local antibacterial and antimonilial protection particularly for conditions such as pruritis ani and vulvae and diaper rash which are representative examples where bacteria and monilia cause or contribute to the disease.

Supplies of Kenacomb Ointment in 5 mg. tubes are now fully available in South Africa.

Further information may be obtained from Squibb Laboratories (Pty.) Ltd., P.O. Box 9975, Johannesburg.

TRASYLOL

FBA Pharmaceuticals (S.A.) (Pty.) Ltd., the sole distributors for Farbenfabriken Bayer AG., Leverkusen, announce the introduction of their product Trasylol, specific for the treatment of pancreatic diseases, and supply the following information:

Trasylol is a highly purified physiological extract which inactivates the proteolytic pancreatic enzyme trypsin and the vasodilatory hormone kallikrein.

Mode of action. Trasylol inactivates the proteolytic pancreatic enzyme trypsin and thereby ensures the termination of the process of autodigestion of pancreatic and other tissue.

Apart from the inactivation of trypsin, Trasylol also causes the shock, one of the most important symptoms of acute pancreatitis, to be diminished in the following manner: The pancreas produces a hormone called kallikrein which has vasodilatory properties and which is contained in that organ in an inactive form. Kallikrein is activated by trypsin when the pancreas becomes damaged as in acute pancreatitis. The vasodilatory effect of the thus activated kallikrein which is released into the circulation is superimposed on the already severely shocked state of the patient suffering from pancreatitis and greatly aggravates his condition to the point where total collapse is threatened. The only means of interrupting this process, which is continuous, is by inactivating trypsin or kallikrein or both. Trasylol is the only known substance which does both. The vasodilatory effect is abolished, the blood pressure rises and the shock is greatly diminished. Trasylol, therefore, protects the patient against the hazards of both the autodigestion by trypsin and the threatening collapse as long as these dangers exist during the acute phase of pancreatic disease. The severe pain caused by the autodigestion by trypsin subsides within a few hours after commencing treatment and the increased urinary diastase values quickly return to normal.

Indications. Trasylol is indicated in all forms of acute pancreatitis and pancreatic necrosis, chronic pancreatitis whenever inactivation of trypsin becomes necessary, in duodenal fistulae, and as a prophylactic in cases of surgery involving the pancreas or the pancreatic area and in all cases where pancreatic complications can be expected.

Contra-indications. No contra-indications are, as yet, known.

Side-effects. Trasylol is extremely well tolerated, even in doses many times those recommended. The only side-effects are nausea and vomiting due to a too rapid intravenous injection.

Dosage. For best results adequate dosage is important and may have to be adjusted according to the severity of the symptoms. Treatment should be initiated by injecting Trasylol slowly (10,000 kallikrein inactivating units) by the intravenous route, followed by continuous i.v. infusion for several days up to 2 weeks.

Packing. Trasylol is supplied in boxes of 10 ampoules of 2 c.c. containing 2,000 kallikrein inactivating units each.

Further information may be obtained from FBA Pharmaceuticals (S.A.) (Pty.) Ltd., P.O. Box 10233, Johannesburg.

MELLERIL

Sandoz Ltd., of Basle, Switzerland, announce the introduction of Melleril, and supply the following information:

Composition. 10 mg., 25 mg., and 100 mg. tablets of thioridazine HCl. Sandoz.

Action and uses. Melleril has a specificity of action on certain brain sites, in contrast to the more 'diffuse' action of other phenothiazines. This is evidenced by minimal antiemetic effect, and minimal extrapyramidal stimulation. Melleril has less 'spill-over' action to other brain areas, hence the virtual elimination of undue sedation, drowsiness or disturbance of the autonomic nervous system. Melleril has virtual freedom from such toxic effects as jaundice, Parkinsonism, skin photosensitivity, skin eruptions, disturbed body-temperature regulation, blood dyscrasias, etc. All these properties mean improved tranquillization with a greater margin of safety in general office practice, in ambulatory psychiatric out-patient clinics, and in hospitalized patients.

Melleril has value in the following indications: Mental and emotional disturbances in the office — anxiety, acute and chronic apprehension and tensien states, psychoneuroses; mental and emotional disturbances in the hospital — pre- and postoperative apprehension and agitation, severe psychoneuroses, agitated psychoses; behaviour problems in children; agitated geriatric patients; intractable pain; and alcoholism.

Administration and dosage. Usual starting dose in non-psychotic patients is 10 or 25 mg. 3 or 4 times a day. Psychotic

patients require 100 mg. 3 or 4 times a day as a starting dose. Dosage must be individually adjusted until maximum therapeutic response is achieved.

Precaution. As with all synthetic drugs, regular blood counts are desirable during protracted treatment.

Contra-indication. Melleril is contra-indicated in any severely depressed or comatose states.

Supplied. Melleril is supplied in 10 mg. tablets in containers of 25 and 100; in 25 mg. tablets in containers of 50, 200 and 1,000; and in 100 mg. tablets in containers of 25, 100 and 1,000.

Further information, samples and literature may be obtained from Sandoz Pharmaceutical Department, P.O. Box 4461, Johannesburg. Sole distributors for South Africa: Alex. Lipworth Limited, P.O. Box 4461, Johannesburg.

MER - 29

Mer-National Laboratories (Pty.) Ltd. announce the introduction of Mer-29, and supply the following information:

Mer-29 is the Merrell brand name for triparanol, a new synthetic organic compound which is the first specific inhibitor of excessive cholesterol biosynthesis.

Pharmacology. Radio-isotope studies in both animals and humans indicate that Mer-29 directly inhibits cholesterol production in the liver, intestines and other tissues. This inhibitory action is partial, and occurs at the final stage of cholesterol biosynthesis. Mer-29 produces substantial reductions in both serum and tissue cholesterol. The cholesterol/phospholipid ratio is significantly reduced, and in animals, studies indicate that Mer-29 produced a fall in alpha- and beta-lipoprotein cholesterol, without affecting serum lipoproteins.

Advantages. Mer-29 offers for the first time, a practical, safe, and effective solution to the problem of hypercholesterolaemia. Mer-29 is the only specific inhibitor of excessive cholesterol biosynthesis, reducing both serum and tissue cholesterol without dietary restrictions. In over 2,000 patients under clinical trial, elevated serum cholesterols were reduced in 85 - 90% of cases. In general, the reduction was proportional to the pretreatment control levels, and the maximum fall was generally obtained after the 4th week of therapy. Increasing the daily dosage did not result in lower cholesterol levels. These findings indicate that Mer-29 will not depress cholesterol levels below those necessary for normal metabolic needs. Because Mer-29 partially inhibits at the final stage of cholesterol biosynthesis — after formation of the steroid nucleus — it does not interfere with adrenal, endocrine, and other vital metabolic functions.

It has been shown that endogenous synthesis accounts for the main source (over two-thirds) of body cholesterol. Low-fat dietary restrictions are thus ineffective in preventing elevated serum-cholesterol levels.

Mer-29 reduces endogenous cholesterol biosynthesis by 36%. This reduction occurs both in serum and tissues, because Mer-29 has been shown to lower the total body miscible pool of cholesterol. The possibility of transferring cholesterol from serum to tissues is thus excluded.

Preliminary findings indicate that after Mer-29 therapy, both objective and subjective improvement has been reported in angina patients using the master 'two-step' exercise-tolerance test. It has been suggested that Mer-29 may improve the adequacy of coronary circulation, and possibly also reduce the cholesterol content of human blood vessels including coronary arteries. Investigations are proceeding.

Side-effects. Extensive, long-term trials have revealed no interference with normal liver and kidney functions, and no blood dyscrasias. Adrenal and endocrine functions are unaltered. An occasional mild gastro-intestinal upset and a skin rash have been reported. A false-positive reaction for albuminuria occurred in a few cases.

Indications. Patients with hypercholesterolaemia; adjunctive therapy for conditions associated with abnormal cholesterol metabolism (coronary-artery disease, generalized atherosclerosis, diabetes, cerebral arteriosclerosis).

Compatibility. Mer-29 is compatible with anticoagulants, vasodilators, and other drugs used in cardiovascular disease.

Caution. Mer-29 should not be used during pregnancy.

Dosage. One 250 mg. tablet once a day before breakfast.

Supplied. Mer-29 is supplied in bottles of 30, light-yellow tablets of 250 mg.

Mer-29 is marketed in South Africa by Mer-National Laboratories (Pty.) Ltd., P.O. Box 6742, Johannesburg, from whom further information may be obtained; and distributed by Westdene Products (Pty.) Ltd., P.O. Box 7710, Johannesburg.

CORRESPONDENCE : BRIEWERUBRIEK

APPROVED MEDICAL AID SOCIETIES

To the Editor: I have read with interest the correspondence which has appeared in your Columns recently requesting the abolition of the Tariff of Fees for Approved Medical Aid Societies and now desire to emphasize certain aspects of the case which up to this juncture have not been mentioned. I write as a layman and I am thus undeniably in the position of being able to present the other side of the case.

I am sure that I can say without fear of contradiction from other people in the same position as myself, that there has been no deterioration in the doctor-patient relationship. On the contrary, I am certain that this relationship has improved since, as a member of an approved medical aid society, both my doctor and I are now spared the embarrassment of attempting to discuss my financial position so that he may assess a fair fee for his services. I *know* that the doctor will be paid a fair fee and the doctor does not need to worry whether the account which he has rendered is too large.

It must be emphasized that this happy position can only be maintained where the medical aid society is an organization which does not seek to make a profit either *directly* or *indirectly* out of an illness.

At the moment, I am happy to say, all the approved medical aid societies fulfil an extremely necessary requirement in our society scheme — they ensure that a patient may send for a doctor when necessary without having to think whether extra expense can be afforded — and they ensure that the doctor is paid a fair fee for his services.

Patient

Cape Town
12 October 1960

SEPARATE EYE HOSPITALS IN SOUTH AFRICA

To the Editor: It was with the greatest interest that we read Dr. Hamelberg's timely address[1] to the Transvaal Sub-Group of the Ophthalmological Society of South Africa stressing the need for separate eye hospitals in Souh Africa.

In Cape Town for some years now we have felt a similar need for a well-equipped eye hospital, standing on its own feet. The Cape Town Sub-Group of the Ophthalmological Society of South Africa, at a meeting held recently, passed a resolution urging the establishment in Cape Town of a teaching eye hospital to serve the Cape Province. A sub-committee was appointed to go into the matter of present facilities and future needs, and prepare figures in support of this. This hospital would not replace existing undergraduate teaching facilities at Groote Schuur and Karl Bremer Hospitals, but would be supplementary to these. It would also serve as a centre for research and advanced or postgraduate teaching.

At present even at these teaching institutions which are 'closed hospitals' the facilities available for special investigation of eye cases, e.g. detachment of the retina, uveitis, pathology and glaucoma, are non-existent or limited. Furthermore, specialized methods of treatment, e.g. corneal grafting, the giant magnet, foreign-body location and so on, are available only at these institutions and not to outside doctors.

An important factor which Dr. Hamelberg did not mention, is the present lack of trained nursing staff for such institutions.

In Cape Town today it is difficult to acquire essential ophthalmic apparatus because these are required in each teaching-hospital department whereas the establishment of a

central eye hospital would obviate this duplication. There is already a measure of support from public bodies for this project and we hope in the near future to put forward a strong case for a separate eye hospital.

J. G. Louw
Chairman, Cape Town Sub-Group
Ophthalmological Society of South Africa (M.A.S.A.)
Medical Centre
Heerengracht
Cape Town
1 October 1960

1. Hamelberg, H. J. (1960): S. Afr. Med. J., **34**, 801.

DR. A. SIMPSON WELLS MEMORIAL FUND

To the Editor: Many friends and admirers of the late Dr. A. Simpson Wells, we feel sure, would like to be associated with us in creating a fund in his memory to further the work which was so dear to him.

In 1917, i.e. 43 years ago, single-handed and with the loyal cooperation of Miss E. M. Hefford, he commenced the training of two or three young Coloured girls in the old St. Monica's Home in Bree Street.

The South African Medical and Dental Council together with the South African Nursing Council agreed that the trainees of the home would be granted recognition provided they fulfilled the existing regulations for the training of midwives.

Thus Dr. Simpson Wells may justly be called the pioneer of the training of Non-European midwives in South Africa.

Today there are hundreds of St. Monica's midwives working amongst their own people in the Union and the Federation.

Those who would like to honour his name are invited to send donations to the Secretary, St. Monica's Home, Lion Street, Cape Town.

S. W. Lavis, *Bishop*
Chairman
E. C. Crichton
Acting Medical Superintendent
St. Monica's Home
Lion Street
Cape Town
4 October 1960

NEED FOR DERMATOLOGISTS

To the Editor: May I be allowed to use your columns to issue an appeal? I should like to ask those colleagues, including interns, who wish to become dermatologists, to get in touch with me.

Year by year this country has stood in an increasing need for young specialists with a modern training in dermatology. Considerable opportunities exist for them, and will continue to exist for some time to come. In a survey published by the Department of Education, we were surprised and pleased to see that at least 26 South African practitioners wished to become dermatologists. We shall be glad to hear from them and from any others with similar aspirations.

Those who are undecided are particularly welcome to approach me, in case we can help them. The Dermatologists' Sub-Group regards it as important if some worthy colleagues can be added eventually to their number by informing and encouraging them.

G. H. Findlay
President, Dermatological Sub-Group (M.A.S.A.)
528 Medical Centre
Pretorius Street
Pretoria
1 October 1960

ABOLITION OF TARIFF OF FEES FOR APPROVED MEDICAL AID SOCIETIES

To the Editor: I shall first relate a few experiences. On being called to a new patient in a new suburb to see a non-White servant, having completed the examination, I was asked to 'write the prescription in my name, and when you render the account put down 3 visits, because that was my arrangement with my doctor in town'. My reply was that normal private fees would be charged. Her husband's medical aid society continues to pay 3 visits for 1, if not to her former family doctor, then to the new one, who has been 'properly educated in attending medical aid society patients'.

Each doctor is naturally jealous of the good understanding he has established with the family, and none is more disappointed when the patient changes his doctor 'at the drop of a hat'. A colleague who has attended a certain family for several years goes on a short holiday — I do his 'locum', and hear from the lady of the house that 'he had the cheek to charge £1 10s 0d. for attendance on her father'. 'I should like you to be our family doctor — if you would charge medical aid society fees for father (who does not belong to a medical aid society)'. She has retained her former family doctor. Each one of us could quote similar instances of intrigue and deception and, provided nothing is unmasked, the medical aid society system will continue to flourish.

In a practice bordering on a larger centre the visiting fee is 15s. An example: The patient's home is exactly 4·9 miles away, and there is only one main arterial road precluding a 'round the block' visit. At a 1s. per mile this costs the doctor 10s., leaving him 5s. for the privilege of practising the art of medicine. Subsequent visits necessitate more than a token history and examination, unlike the token 5s. nett. Herein lies the cause of overvisiting. In normal private practice the doctor shows the patient the courtesy of considering the patient's pocket, and will shorten the number of visits to retain the patient's *bona fides*. Now some may argue that it is only a general practitioner's humanitarian service and why be so mean as to earn money out of human illness! Therefore let 5s., and not the patient, be the 'visit-regulator'. However, finally the account for the services is rendered, and comes the turn of the 'witch-hunter' — the board that assesses the number of visits for a specific domestic (or hospitalized) illness and the general practitioner may even lose his 5s. nett.

Many a patient, these days, accuses the doctor of overcharging in terms of the fixed 'cut-price' tariff. This reduces the fee by 25 - 40% in different regions of the country, and the general practitioner is expected to survive as a unit rendering an essential service to a modern civilized community. Small wonder, then, that the patient-doctor relationship deteriorates daily. Abolish the Tariff and restore the doctor's dignity!

Non-Tariff Adherent

4 October 1960

PRINTING ON X-RAY ENVELOPES

To the Editor: My Committee would appreciate your publishing the following important notice to radiologists in both official languages, in the next issue of the *Journal:*

A ruling from the South African Medical and Dental Council has been obtained in this respect which states the provisions of ethical rule 1 (5) should be observed in regard to printing on X-ray envelopes.

Rule 1 (5) does not allow the printing on envelopes of any information other than the practitioner's name without his title or any qualification, and a return address in case of non-delivery.

The South African Medical and Dental Council have recently clarified this ruling as follows:

'It is permissible to print the words "X-ray films with care — do not fold" or words similar to that effect.

'Although it is permissible to print the doctor's name and address in case of non-delivery, it is not permissible to print the word "doctor" or "radiologist" or any qualification.'

(„Dit is toelaatbaar om die woorde „röntgenfoto's versigtig — moenie vou nie" of woorde wat dieselfde beteken te druk.

„Alhoewel dit toelaatbaar is om die dokter se naam en adres te druk in gevalle waar aflewering nie plaasvind nie, is dit nie toelaatbaar om die woord „dokter" of „röntgenoloog" of enige kwalifikasie te druk nie.')

C. Komins
Hon. Secretary
Radiological Society of South Africa (M.A.S.A.)
1 Lister Buildings
Jeppe Street
Johannesburg
6 October 1960